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POSTOPERATIVE LUNG ABSCESS

AN EXPERIMENTAL STUDY *

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I. INTRODUCTION

TYPES OF LUNG ABSCESS

Pulmonary suppuration, though long a matter of much clinical interest, remains one of the conditions of which the underlying etiologic factors are still in dispute. Several types of lung abscesses seem to occur, each of which may develop from quite different causes. With our present knowledge, classification from the pathologic point of view seems wisest. Aschner [1] groups the several types as follows:

1. Bronchiectasis.
2. Bronchiectatic abscess.
3. Suppurative pneumonitis.
4. Extrabronchial abscess.

Among the more common clinical forms of lung abscess is that which is now recognized as developing subsequent to operation. This recent recognition emphasizes the importance of studying and classifying postoperative lung abscess as a separate clinical entity. The considerable proportion of such abscesses following the operation of tonsillectomy has led the otolaryngologists to study, report on and investigate this condition from both the clinical and laboratory sides. The majority of such reports and investigations conclude that postoperative lung abscess is due to aspiration during the operation. This is in spite of the fact that lung abscess has never been satisfactorily reproduced by the experimental instillation of infected material and foreign bodies by way of the

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1. Aschner, P. W.: The Pathology of Lung Suppuration, Ann. Surg. **75**:321 (March) 1922.

trachea, and also in spite of the fact that others, notably Jackson,[2] interested in bronchoscopic work report that typical lung abscess is rare with the lodgment of foreign bodies even deep in the air passages.

In an attempt to search for the mechanism that would explain postoperative lung abscess, we repeated the experimental introduction of infected materials by way of the trachea. We were unsuccessful as had been our predecessors. This led to the necessity for a different explanation, and, being conversant with the more recent theory that embolism from the wound may well be the cause of other postoperative pulmonary complications,[3] it seemed possible that such a mechanism might well occur in relation to this single group. From the first our experiments were satisfactory, and we have developed a uniformly successful method[4] for the production of lung abscess by freeing septic emboli into the vascular bed.

In the etiologic discussions of the older writers, the relationship that operative measures hold toward the production of lung abscess was not considered. Mention is made of septic emboli causing pulmonary abscess, but these were frequently multiple, constituting a part of the picture of a pyemia which may or may not have been preceded by an operation. That operative procedures play a significant part in the etiologic rôle of lung abscess is now recognized. A summary of the articles dealing with this complication reveals two important facts: (1) the increasing frequency of the condition postoperatively, and (2) the apparent marked increase following tonsillectomy. The increase in frequency is due in part to the increased recognition for which the roentgen ray is in a large measure responsible. The greater percentage after tonsil operations can also be attributed to a more careful anamnesis.

FREQUENCY OF OCCURRENCE

Ever since 1912 when Richardson[5] reported the first case of lung abscess following tonsillectomy, the percentage incidence of postoperative lung abscess in the recorded cases has reached a progressively higher figure. The following year one lung abscess was found by Bassim[6] among nineteen pulmonary complications

2. Jackson, C.: Suppurative Diseases of the Lung Due to Inspirated Foreign Body Contrasted with Those of Other Etiology, Surg. Gync. Obst. **42**:305 (March) 1926.

3. Cutler, E. C., and Hunt, A. M.: Postoperative Pulmonary Complications, Arch. Surg. **1**:114 (July) 1920; Postoperative Pulmonary Complications, Arch. Int. Med. **29**:449 (April) 1922. Cutler, E. C.: The Etiology of Postoperative Pulmonary Complications, S. Clin. N. Amer. **2**:935 (Aug.) 1922.

4. Holman, E.; Weidlein, I. F., and Schlueter, S. A.: A Method for the Experimental Production of Lung Abscess, Proc. Soc. Exper. Biol. & Med. **23**: 266, 1926.

5. Richardson, C. W.: Tonsillectomy with Consideration of Its Complications, Washington M. Ann. **12**:2, 1912.

6. Bassim: Les Complications broncho-pulmonaires consecutives a l'adenoidectomie et a l'amygdalectomie, Paris thesis, 1913, no. 181.

following tonsillectomy and adenotomy. In 1914 Scudder [7] reported sixteen cases of lung abscess treated by surgery. Three occurred postoperatively, one after a nasal septum operation and two after duodenal ulcer operations. The second case of lung abscess following tonsillectomy in American literature was reported by Manges [8] in 1915. The following year Coakley [9] reported a similar case, Manges [10] compiled nine cases and Richardson [11] reported three cases, all occurring after tonsillectomy. In 1917 Frank [12] compiled three such cases and Tewksbury [13] published two cases, one of which followed tonsillectomy and the other a nasal operation. Tewksbury employed artificial pneumothorax and was the first to institute this procedure as a therapeutic measure for the acute abscess. The following year he submitted his results on ten acute abscesses,[14] all following nose and throat operations. From time to time numerous individual cases have been recorded, but only the larger series will be dealt with further.

In a study of 100 cases of lung abscess by Wessler [15] twenty-six followed operation, twenty-one of which were posttonsillectomy. Fifteen additional cases were reported by Wessler and Schwarz,[16] five of which followed tonsillectomy. Fisher and Cohen [17] compiled seventy-six cases after tonsillectomy. Of thirty-eight cases treated by bronchoscopy by Lynah,[18] nine occurred after operation. Moore [19] compiled 202 abscesses occurring in approximately 450,000 tonsillectomies. Prosser [20] found eighteen cases among the records of the medical division of the University of Pennsylvania Hospital, six of which followed some operative pro-

7. Scudder, C. L.: A Report of the Cases of Lung Abscess at the Massachusetts General Hospital Clinic, Boston M. & S. J. **171**:523, 1914.

8. Manges, M.: Nontuberculous Pulmonary Suppurations, J. A. M. A. **64**: 1554 (May 8) 1915.

9. Coakley, C. G.: Lung Abscess Following Tonsillectomy, Laryngoscope **26**: 1008 (July) 1916.

10. Manges, M.: The Occurrence of Abscess of the Lung After Tonsillectomy, Am. J. Surg. **30**:78 (March) 1916.

11. Richardson, C. W.: Abscess of the Lung Following Operation on the Tonsils and Upper Air Tract, Laryngoscope **26**:1001 (July) 1916.

12. Frank, I.: On Lung Abscess as a Sequel to Tonsillectomy, Laryngoscope **27**:474 (June) 1917.

13. Tewksbury, W. D.: Acute Pulmonary Abscess Treated with Artificial Pneumothorax, J. A. M. A. **68**:770 (March 10) 1917.

14. Tewksbury, W. D.: Treatment of Nontuberculous Lung Abscess with Pneumothorax, J. A. M. A. **70**:293 (Feb. 2) 1918.

15. Wessler, H.: Lung Abscess and Bronchiectasis: A Clinical and Roentgenological Study of 100 Cases, Am. J. Roentgenol. **6**:161 (April) 1919.

16. Wessler, H., and Schwarz, H.: Abscess of Lungs in Infants and Children, Am. J. Dis. Child. **19**:137 (Feb.) 1920.

17. Fisher, L., and Cohen, A. J.: Pulmonary Abscess in Adults Following Tonsillectomy Under General Anesthesia, J. A. M. A. **77**:1313 (Oct. 22) 1921.

18. Lynah, H. L.: Bronchoscopic Studies of Pulmonary Abscess, J. A. M. A. **77**:1548 (Nov. 12) 1921.

19. Moore, W. F.: Pulmonary Abscess: An Analysis of Two Hundred and Two Cases Following Operative Work About the Upper Respiratory Passages, J. A. M. A. **78**:1279 (April 29) 1922.

20. Prosser, W. O. H.: A Review of Eighteen Cases of Pulmonary Abscess, Mil. Surgeon **51**:37 (July) 1922.

cedure. In Lockwood's [21] series of fifty-three cases, 50 per cent were postoperative. Whittemore's [22] statistics on 100 cases reveal sixty-six (66 per cent) as having been preceded by some operation on the upper respiratory tract. Sante [23] in a serial radiographic study of forty-five cases found six to be postoperative; two followed tonsillectomy and four followed laparotomy. There were four postoperative lung abscesses in the twelve cases studied by Lewald and Green.[24] Lockwood [25] in 1923 compiled 208 cases following tonsillectomy. Homan's [26] study comprises twenty-three cases at the Peter Bent Brigham Hospital. Ten occurred after nose and throat surgery and three following abdominal operations. Heuer and MacCready,[27] in summarizing the sixty-two lung abscesses in the Johns Hopkins Hospital records, found that sixteen occurred after operation, four of which followed tonsillectomy. Glowacki [28] studied ninety cases from a number of St. Louis hospitals during a period of five years. An analysis of only twenty-eight is given. Singer and Graham [29] reported thirty-four cases; eleven (32 per cent) followed an operation. Mackenzie [30] published a report based on 169 cases from Bellevue Hospital, New York, during the years 1918-1922, eleven of which followed tonsillectomy. The total number following operation is not given. In the hundred cases reported by Lambert and Miller,[31] twenty-five followed operation, thirteen of which followed tonsillectomy. In a large series of 692 cases observed at the Mayo Clinic by Hedblom,[32] 146 (21 per cent) followed an operation. Of these 146 cases forty-eight (33 per cent) occurred after tonsillectomy. Winner [33] reported twenty-two cases; nine were postoperative. In an analysis of 227 cases by Lord,[34] ninety-six (42 per cent) followed a surgical procedure. Greer [35] analyzed

21. Lockwood, A. L.: Abscess of the Lung, Surg. Gynec. Obst. **35**:461 (Oct.) 1922.

22. Whittemore, W.: The Etiology and Treatment of Nontuberculous Pulmonary Abscess, Surg. Gynec. Obst. **38**:461 (April) 1924.

23. Sante, L. R.: A Study of Lung Abscess by Serial Radiographic Examination, J. Radiol. **4**:183 (June)1923.

24. Lewald, L. T., and Green, N. W.: The Differential Diagnosis Between Tuberculosis and Lung Abscess, Arch. Surg. **6**:303 (June) 1923.

25. Lockwood, A. L.: Lung Abscess, Arch. Surg. **6**:314 (Jan.) 1923.

26. Homans, J.: The Etiology and Clinical Features of Lung Abscess, Boston M. & S. J. **188**:577 (April 19) 1923.

27. Heuer, G. J., and MacCready, P. M.: Lung Abscess, Arch. Surg. **6**:337 (Jan.) 1923.

28. Glowacki, B. F.: Pulmonary Abscess: A Study of Ninety Cases, Laryngoscope **33**:153 (Feb.) 1923.

29. Singer, J. J., and Graham, E. A.: A Study of Thirty-Four Cases of Abscess of the Lung, J. A. M. A. **81**:193 (July 21) 1923.

30. Mackenzie, L. B.: Lung Abscess, M. J. & Record **119**:191 (Feb. 20) 1924.

31. Lambert, A. V. S., and Miller, J. A.: Abscess of the Lung, Arch. Surg. **8**: 446 (Jan.) 1924. Miller, J. A., and Lambert, A. V. S.: The Treatment of Abscess of the Lung, Am. J. M. Sc. **171**:81 (Jan.) 1926.

32. Hedblom, C. A.: The Surgical Treatment of Acute Pulmonary Abscess and Chronic Pulmonary Suppuration, J. A. M. A. **83**:1577 (Nov. 15) 1924.

33. Winner, P. S.: A Study of Twenty-Two Cases of Lung Abscess, Illinois M. J. **47**:267 (April) 1925.

34. Lord, F. T.: Certain Aspects of Pulmonary Abscess from an Analysis of 227 Cases, Boston M. & S. J. **192**:785 (April 23) 1925.

35. Greer, A. E.: Lung Abscess: Thirty-Three Cases, Am. J. M. Sc. **169**: 345 (March) 1925.

thirty-three cases; twelve (36 per cent) followed operation. In a review of thirty-two cases treated bronchoscopically, Myerson[36] found that ten (31 per cent) followed operation. In a more recent article than that of Hedblom,[32] Lemon[37] stated that fifty-three cases of posttonsillectomy abscess had been observed at the Mayo Clinic. Kernan,[38] in a report before the New York Society for Thoracic Surgery, stated that of the seventy-nine cases treated by him bronchoscopically twenty-five (31 per cent) followed operation. An analysis of the ten cases reported by Eggers[39] reveals two (20 per cent) as having followed operations.

The larger reported series comprising 1,908 cases of lung abscess have been compiled and placed in table 1. Of these 1,908 lung abscesses, 515, or 29.6 per cent, have been classed as postoperative complications,

TABLE 1.—*Incidence of Lung Abscess Following Operation*

Author	Number of Cases	Following Operation		Number After Tonsillectomy	Per Cent
		Number	Per Cent		
Scudder	16	3	19	0	0
Wessler	100	26	26	21	21
Wessler and Schwartz	15	5	33	5	33
Lynah	38	9	23	8	21
Prosser	18	6	33	2	11
Lockwood	53	23	43	16	30
Whittemore	100	66	66	48	48
Sante	45	6	13	2	4
Lewald and Green	12	4	33	3	25
Homans	23	13	57	7	30
Heuer and McCready	62	16	26	4	6
Glowacki	28	2	7	2	7
Singer and Graham	34	11	32	8	24
Mackenzie	169	..	..	11	7
Lambert and Miller	100	25	25	13	13
Hedblom	692	146	21	48	7
Winner	22	9	41	7	32
Lord	227	96	42	49	21
Greer	33	12	36	7	21
Meyerson	32	10	31	6	19
Kernan	79	25	31	..	..
Eggers	10	2	20	1	10
	1,908	515	29.61	268	14.61

an amazing number. It will also be seen that the operation of tonsillectomy contributes in no small measure as a causative factor, being responsible for 14.6 per cent of the total. Statistics vary a great deal regarding the frequency of lung abscess after tonsillectomy. Lyman[40] states that 20,000 tonsillectomies have been performed under nitrous oxide anesthesia in the medical department of Washington University without the development of a single postoperative lung abscess. Out of

36. Myerson, M. C.: The Bronchoscopic Treatment of Lung Abscess, Surg. Gynec. Obst. **41**:573 (Nov.) 1925.

37. Lemon, W. S.: Clinic on Abscess of Lung: Case of Posttonsillectomy Pulmonary Abscess, Arch. Surg. **10**:583 (Jan.) 1925.

38. Kernan, J. A., quoted by Eggers, C.: Lung Abscess Complicated and Hidden by Empyema, Arch. Surg. **12**:338 (Jan.) 1926.

39. Eggers (footnote 38).

40. Lyman, H. W.: Relation of Nose and Throat Operations to Lung Abscess, J. Missouri M. A. **20**:418 (Dec.) 1923.

the 20,000 tonsillectomies performed at the Mayo Clinic, Hedblom [32] could find only two occurring after the operation. Data obtained from the questionnaires sent out by Moore [19] revealed that 202 cases occurred in approximately 450,000 tonsillectomies, an average of one abscess in every 2,500. Two lung abscesses were observed by Herb [41] in a series of 12,045 tonsil and adenoid operations under general anesthesia. In the statistics obtained by Glowacki [28] from a number of St. Louis hospitals one abscess occurred for every 358 tonsillectomies, while Keiper [42] makes the statement that out of every 781 tonsillectomies one lung abscess is encountered. The source of these figures is not given.

Deductions based on this inadequate statistical data would be quite misleading. That the operation of tonsillectomy has aided considerably in increasing the number of postoperative lung abscess cases is unquestionably true, but the condition attains importance only in a large operative series. Many consider the lesion to be a common complication after tonsillectomy, but the combined figures would seem to indicate that this is a mistaken conception. Furthermore, in view of the vast number of tonsil operations performed every year, it is evident that such a complication is rather infrequent.

ETIOLOGY

In the previous etiologic discussion of postoperative lung abscess, opinions have been formed chiefly from clinical study and observation, and scant attention has been paid to the experimental work already performed in this particular field. Moreover, the elaboration and application of suitable therapy has thus far received major attention. It is highly desirable that the etiology of any known disease be substantiated by experimental investigation. Laboratory studies designed to contribute something toward a solution of the causative factors of postoperative lung abscess should be of considerable significance, and, from the standpoint of therapy, advantages from such a study may perhaps be efficaciously applied.

Interest in the entire group of postoperative pulmonary complications naturally incited those who were cognizant of the often dire sequelae to propound certain theories explaining the pathways pursued by the causative agent to the lung. Three possible courses along which infection might travel were recognized, viz., (1) by direct lymphatic extension from the operative area; (2) by way of the bronchi from infected material aspirated during or immediately after the operation,

41. Herb, I. C.: Postoperative Lung Complicatians, J. A. M. A. **79**:339 (July 29) 1922.

42. Keiper, G. F.: The Tonsil Question Up to Date, Laryngoscope **31**:777 (Oct.) 1921.

and (3) by way of the blood stream in the form of septic emboli from the thrombosed vessels of the operative field. In the vast amount of rapidly accumulating literature dealing with postoperative lung abscess the same views have been applied and repeatedly discussed.

That the lymphatic route is a possibility has been shown by the anatomic studies of Sabin [43] and Miller.[44] Not a few writers, notably Clendening,[45] gave this view considerable support. The other two views have, however, caused the greatest amount of discussion.

The early investigative work of Hoelscher [46] and Kelly [47] and more recently that of Lemon [48] and Myerson [49] is sufficient experimental evidence that aspiration of oral contents occurs during anesthesia, but the corollary that this means lung infection does not necessarily follow. That aspiration probably occurs in a high percentage of patients undergoing operations under general anesthesia is an accepted fact. If we admit such a possibility, a greater frequency of lung complications should be expected, even in numbers that would exceed the statistics published by Cutler and Hunt.[3] Such is not the case. In spite of badly administered anesthesia, pulmonary complications fail to develop, and in the better clinics where skilled anesthetists are now employed these complications continue to occur.

The presence of specific bacteria has been taken by some investigators as a factor of major importance. Group IV pneumococcus has been found repeatedly in normal mouths [50] and the isolation of these organisms from postoperative pneumonia patients by Whipple [51] and Cleveland [52] con-

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43. Sabin, F. R.: The Method of Growth of the Lymphatic System, Science, New York **44**:145, 1916.

44. Miller, W. S.: Some Essential Points in the Anatomy of the Lung, Am. J. Roentgenol. **4**:269 (June) 1917; The Vascular Supply of the Pleura Pulmonalis, Am. J. Anat. **7**:389, 1908.

45. Clendening, L.: The Cause of Abscess of the Lung After Tonsillectomy, J. A. M. A. **74**:941 (April 3) 1920; Abscess of the Lung, Laryngoscope **32**:128 (Feb.) 1922.

46. Hoelscher, R.: Experimentelle Untersuchungen über die Entstehung der Erkrankungen der Luftwege nach Aethernarkose, Arch. f. klin. Chir. **57**:175, 1898.

47. Kelly, R. E.: Anesthesia by the Intratracheal Insufflation of Ether, Brit. M. J. **2**:112 (July 20) 1912; The Intratracheal Insufflation of Ether, Brit. M. J. **2**:617 (Sept. 14) 1912.

48. Lemon, W. S.: Aspiration: Experimental Study, Arch. Surg. **12**:187 (Jan.) 1926.

49. Myerson, M. C.: Bronchoscopic Observations on the Cough Reflex in Tonsillectomy under General Anesthesia, Laryngoscope **34**:63 (Jan.) 1924.

50. Stillman, E. G.: A Contribution to the Epidemiology of Lobar Pneumonia, J. Exper. Med. **24**:651 (Dec.) 1916.

51. Whipple, A. O.: A Study of Postoperative Pneumonitis, Surg. Gynec. Obst. **26**:29 (Jan.) 1918.

52. Cleveland, M.: Further Studies in Postoperative Pneumonitis, Surg. Gynec. Obst. **28**:282 (March) 1919.

stitutes an argument for the aspiration mechanism. A few investigators, Blake and Cecil [53] and Kline and Winternitz,[54] have succeeded in producing pneumonia experimentally by intrabronchial insufflation but only when a large number of organisms were used. In contrast to these results the researches of Stillman [55] are of interest. Experiments were carried out with mice exposing them to an atmosphere containing cultures of bacteria in the form of fine mist. Pneumococcus, *Streptococcus hemolyticus, Bacillus influenzae,* and staphylococcus readily penetrated into the lower respiratory tract. Pneumococci usually disappeared within a few hours and gave rise to no infection even if the mice were chilled. The experiments fail to shed any light on the exact mode of natural infection of the lung with pneumococci. "They indicate that even in so susceptible an animal as the mouse other factors than the presence of pneumococci in the lung are necessary for infection."

It is difficult to base conclusions on such conflicting evidence, and there remains considerable doubt in the opinion of many that aspiration is the chief cause of postoperative pneumonia. That postoperative lung abscess is the result of aspired infectious material is even more difficult to understand. Here the pathologic lesion is a well circumscribed, limited nidus of infection which continues to enlarge by destroying adjacent parenchymatous tissues, including bronchioles, and finally ruptures into a bronchus adequate in size for its evacuation, or directly into the pleural cavity. As will be shown later, attempts to duplicate lung abscess by intrabronchial measures in animals have proved futile with all investigators. This may be due to the fact that the postoperative lesion was assumed to be a bronchiectatic lung abscess, whereas in reality the condition probably involves originally the parenchymatous tissues and, therefore, has its beginning outside of the bronchial tree. If it could be proved that the process started in this fashion, the manner of its production would be simple. The source of the infection would then obviously have been by way of the blood stream. That lung abscess is derived by such a mechanism can only be proved by accurate pathologic studies through the various evolutionary stages from its incipiency. Since this is almost an impossibility in clinical cases, final conclusive evidence will probably never be realized.

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53. Blake, F. G., and Cecil, R. L.: Studies on Experimental Pneumonia; Production of Pneumococcus Lobar Pneumonia in Monkeys, J. Exper. Med. **31**:403 (Oct.) 1920.

54. Kline, B. S., and Winternitz, M. C.: Studies upon Experimental Pneumonia in Rabbits; The Production of Lobar Pneumonia, J. Exper. Med. **21**:304, 1915.

55. Stillman, E. G.: The Presence of Bacteria in the Lungs of Mice Following Inhalation, J. Exper. Med. **38**:117 (Aug.) 1923.

Since a knowledge of the initial pathologic process cannot be obtained in this manner, and in order to show that the production by embolism constitutes a true premise, it is necessary to interpret clinical symptoms and physical signs correctly. Application of these findings in terms of a pathologic process will then furnish sufficient evidence from which justifiable deductions can be drawn as regards the mechanism responsible for the mode of development. If the infective agent is aspired, early or immediate signs and symptoms will be manifested, in all probability during the first postoperative day. Following a variable incremental period coexistent with the abscess, the period of resolution commences either by lysis from gradual absorption or by crisis from rupture and evacuation into a bronchus. If the infective agent is conveyed to the lung by the blood stream, the abscess will begin at a time correspondent with the dislodgment of the embolus from the source of infection. When abscess results in this manner, it follows the operative procedure after a variable length of time, although the breaking loose of a thrombus is usually considered a later event. Symptoms resulting from such a mechanism will of necessity usually exhibit themselves abruptly. There is abundant reference material in the previous writings and numerous case reports which, when carefully studied from a symptomatic point of view, places these lung abscesses in the true parenchymatous class, and they therefore represent lesions resulting from emboli and not from aspiration.

Adequate support of the embolic theory is furnished in a number of early writings, notably those of Otte,[56] Homans,[57] Gebele,[58] Gottstein,[59] Henle [60] and Mikulicz.[61] The latter three authors were able to select a greater percentage of pulmonary complications in patients in whom local anesthesia was employed than in those in whom a general anesthetic was used. Ranzi [62] placed great stress on the frequency of postoperative lung embolism and considered lung infarctions and empyema the result of emboli. The importance of pulmonary embolism and infarction as

56. Otte, A.: Ueber die Postoperativen Lungen Komplikationen und Thrombosen nach Aethernarkosen, München. med. Wchnschr. **54**:2473 (Dec. 10) 1907.

57. Homans, J.: Postoperative Pulmonary Complications, Bull. Johns Hopkins Hosp. **20**:128, 1909.

58. Gebele: Ueber Embolische Lungenaffektionen nach Bauchoperationen; Eine klinisch-experimentelle Studie, Beitr. z. klin. Chir. **43**:251, 1904.

59. Gottstein, G.: Erfahrungen über lokale Anästhesie in der Breslauer Chirurgischen Klinik, Arch. f. klin. Chir. **57**:409, 1898.

60. Henle: Die Methoden der Schmerzbetäubung und ihre gegenseitige Abgrenzung, Verhandl. d. deutsch. Gesellsch. f. Chir. **30**:240, 1901.

61. Mikulicz, J.: Die Methoden der Schmerzbetäubung und ihre gegenseitige Abgrenzung, Verhandl. d. deutsch. Gesellsch. f. Chir. **30**:560, 1901.

62. Ranzi, E.: Ueber postoperativen Lungencomplikationen embolischer Natur, Arch. f. klin. Chir. **87**:380, 1908.

the real etiologic factors in postoperative pulmonary complications is further emphasized by Boman [63] and Lockhart-Mummery.[64] Bingold [65] is of the opinion that pulmonary emboli frequently do not display the symptomatology the textbooks teach. There may be only localized pains in the chest followed by hemorrhagic sputum, or the formation of an abscess. Mason [66] in his experimental work on pulmonary embolism and thrombosis states that many of the so-called postoperative pneumonias are embolic processes. Capelle [67] attempts to demonstrate the great frequency of embolism subsequent to operation. He recognizes two types of emboli, the large fatal type and the small type resulting in pleuropneumonic signs which may give rise to a bloody sputum and which usually exhibit pleuritic pain and a friction rub as the first sign. The onset occurs from six to fifteen days after operation, whereas aspiration pneumonia would take place earlier. Similar observations and descriptions of thrombus formation and dislodgment were made by McCann,[68] Ochsner and Schneider,[69] Hampton and Wharton [70] and Rupp.[71] DeQuervain [72] makes this statement:

> The most important causes of death—and really three fourths of the true postoperative deaths are due to lung complications—are emboli, pneumonia and lung gangrene. The danger of these complications depends more on the extent of operation than on the question of anesthesia. Even after local anesthesia we have seen embolic processes and pneumonia. Perhaps there will always be an opportunity here to improve our method of procedure. The statistics from our clinic confirm the old conception that a good share of cases which suffer, following

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63. Boman, P. G.: Postoperative Pulmonary Complications, Minnesota Med. **8**:517 (Aug.) 1925.

64. Lockhart-Mummery, P.: Postoperative Pulmonary Embolism, Brit. M. J. **2**:850 (Nov. 8) 1924.

65. Bingold, K.: Symptoms of Pulmonary Embolism, München. med Wchnschr. **72**:1237 (July 24) 1925.

66. Mason, E. C.: Blood Coagulation; the Production and Prevention of Experimental Thrombosis and Pulmonary Embolism, Surg. Gynec. Obst. **39**:421 (Oct.) 1924.

67. Capelle, W.: Einiges zur Frage der Postoperativen Thromboembolie, Beitr. z. klin. Chir. **119**:485, 1920.

68. McCann, F.: Suggestions for the Prevention of Postoperative Thrombosis and Embolism, Brit. M. J. **1**:277 (March 9) 1918.

69. Ochsner, A. J., and Schneider, C. C.: Fatal Postoperative Pulmonary Thrombosis, Ann. Surg. **72**:91 (July) 1920.

70. Hampton, H., and Wharton, L. R.: Venous Thrombosis, Pulmonary Infarction and Embolism Following Gynecological Operation, Bull. Johns Hopkins Hosp. **31**:95 (April) 1920.

71. Rupp, A.: Postoperative Thrombosis and Pulmonary Embolism, Arch. f. klin. Chir. **115**:672 (March) 1921.

72. DeQuervain, E.: A Consideration of the Relative Merits of Resection and Gastro-Enterostomy in the Treatment of Gastric and Duodenal Ulcer, Surg. Gynec. Obst. **34**:1 (Jan.) 1922.

operations upon the stomach, with so-called pneumonia are in reality suffering from a process embolic in nature.

That faults in operative technic, such as infection, careless transfixion of pedicles, massive ligation of blood vessels and rough use of retractors, are conducive to pulmonary embolism is pointed out by Heard [73] and Delore.[74] Cutler and Hunt [8] have probably contributed more toward a better understanding and clearer conception of the importance of embolism in postoperative pulmonary complications than any other workers in this field. Their belief that an embolus from the operative field causes the majority of such complications is being corroborated more and more by other observers.

Conclusions have been reached by many writers that the anesthetic employed is of minor importance. In gastric surgery Grégoire [75] has experienced pulmonary complications in 9 per cent of his cases in which local anesthesia was used. Mandl [76] found that after goiter operations lung complications were more frequent after general than after local anesthesia, but that after hernia operations the reverse was true. In Moore's [19] 202 cases of abscess, thirty-nine followed local anesthesia. The two lung abscess cases after tonsillectomy reported by Porter [77] followed local anesthesia. Two similar cases are reported by Simpson and Noah.[78] Metastatic abscesses of the lungs and secondarily of the liver following tonsillectomy under local anesthesia have been observed in three cases at necropsy by Warthin.[79] In the clinics of Gottstein,[59] Henle [60] and Mikulicz [61] there were more lung complications with local than with general anesthesia. It would seem that such evidence strengthens the embolic theory considerably.

Recently there has been considerable discussion concerning the etiologic factors of posttonsillectomy lung abscess. In a census of recent writers, forty declare themselves in favor of aspiration while only ten favor embolism as the direct cause of lung abscess. The most ardent supporters of the aspiration theory, Moore,[19] Lord,[34] Whitte-

73. Heard, J. E.: Postoperative Pulmonary Embolism, New Orleans M. & S. J. **76**:451 (April) 1924.

74. Delore, X.; Michon, L., and Pollosson, E.: Des Complications Pulmonaires au Cours de la Chirurgie Gastrique, Presse méd. **32**:762 (Sept. 20) 1924.

75. Grégoire, R.: Les Accidents Pulmonaires dans la Chirurgie Gastrique, Bull. et mém. Soc. nat. de Chir. **50**:830, 1924.

76. Mandl, F.: Postoperative Lung Complications, Wien. klin. Wchnschr. **34**: 214 (May 5) 1921.

77. Porter, W. B.: Pulmonary Abscess Following Tonsillectomy Under Local Anesthesia, Virginia M. Monthly **47**:606 (March) 1921.

78. Simpson, J. R., and Noah, H. G.: Report of Two Cases of Lung Abscess Following Tonsillectomy Under Local Anesthesia in Tubercular Subjects, Pennsylvania M. J. **23**:332 (March) 1920.

79. Warthin, A. S., quoted by Frank (footnote 12).

more,[22] Richardson,[80] Tewksbury,[81] Manges,[10] Coakley,[9] Myerson,[82] Hedblom,[32] Pilot and Davis,[83] Glowacki,[28] Chipman,[84] Sante,[23] Meyer,[85] Lemon [48] and Lockwood,[21] base their argument on the following: (1) the common association with general anesthesia; (2) the experimental evidence that aspiration occurs; (3) the high incidence after tonsillectomy and other operations on the upper respiratory tract, and (4) the frequency of lower lobe involvement.

Those who favor embolism, Cutler and Hunt,[3] Homans,[26] Fetterolf and Fox,[86] Porter,[77] Fisher and Cohen,[17] Mackenzie,[30] Lukens,[87] Simpson and Noah,[78] Bevan [88] and Frank,[12] contend that the following facts must be borne in mind: (1) the frequent development after local anesthesia; (2) the high percentage following septic operations or operations performed in potentially infected fields; (3) the greater percentage of occurrence after procedures in mobile operative areas, and (4) the often late development of the symptoms.

The factor of anesthesia and the fact that aspiration occurs have already been sufficiently dwelt on. The comparatively larger number of operations on the upper respiratory tract would naturally predispose to a greater number of pulmonary complications and would, therefore, not constitute a strong argument for the aspiration adherents. We agree with Homans [26] that the aspirating hypothesis is not required to explain the frequency of lower lobe involvement, especially the right. The same percentage holds for lobar pneumonia, infarction, foreign body and embolus localization. The fact that bronchial communications exist with lung abscess is not proof of the aspiratory origin. As the destructive process advances, bronchioles and larger bronchi will gradually be

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80. Richardson, C. W.: Abscess of the Lung and the Method of Prevention in Nasopharyngeal Surgery, Ann. Otol. Rhin. & Laryng. **31**:960, 1922.

81. Tewksbury, W. D.: Acute Pulmonary Abscess, New York M. J. **110**:849 (Nov. 22) 1919.

82. Myerson, M. C.: Lung Abscess Following Tonsillectomy: A Contribution to the Etiology, Arch. Otolaryng. **1**:137 (Feb.) 1925.

83. Pilot, Isadore; and Davis, D. J.: Studies in Fusiform Bacilli and Spirochetes: IX, Their Rôle in Pulmonary Abscess, Gangrene and Bronchiectasis, Arch. Int. Med. **34**:313 (Sept.) 1924.

84. Chipman, C. N.: Relation of Anesthetic to Pulmonary Abscess Following Nose and Throat Surgery, J. A. M. A. **79**:539 (Aug. 12) 1922.

85. Meyer, Willy: Observations on Lung Suppuration and Its Treatment, Arch. Surg. **6**:361 (Jan.) 1923.

86. Fetterolf, G., and Fox, H.: The Reaction of the Paratonsillar Tissues to Tonsillectomy: A Study in the Etiology of Posttonsillectomy Pulmonary Abscess, Am. J. M. Sc. **166**:802 (Dec.) 1923.

87. Lukens, R. M.: Pulmonary Abscess Following Tonsillectomy: A Cure by Bronschoscopic Drainage, S. Clin. N. Amer. **4**:70 (Feb.) 1924.

88. Bevan, A. D.: Abscess of the Lung—An Important Sequel to Tonsillectomy, S. Clin. **2**:921 (Oct.) 1918.

included regardless of whether the abscess originally started intrabronchially or extrabronchially. The time at which the abscess will rupture into a bronchus of sufficient size for evacuation or directly into the pleural cavity is dependent on its anatomic position. A spontaneous occurrence of the latter two events has been described by Hartwell,[89] and Eggers [39] makes the suggestion that rupture into the pleural cavity may be considered as a favorable outcome. Many writers admit that the aspiration of oral or pharyngeal content is not responsible for the ensuing complication per se but that other contributing factors, such as preexisting lung disease, the irritant action of the anesthetic, old age and debility, play an important part. Myerson [82] concludes that the failure of expulsion resulting from damage to the bronchial and lung elements by previous lung infections is the important factor.

The convincing strength of such arguments must be conceded. Based on reliable experimental and clinical evidence, our convictions, however, lead us to believe that embolism is the chief factor. The experimental demonstration by Fetterolf and Fox [86] that thrombi, frequently septic, exist in the tonsillar sinuses, and that dislodgment can easily occur from muscular action, furnishes logical evidence regarding the etiology of posttonsillectomy lung abscess. Mackenzie [30] expresses similar views. The application of suction and the improvement in the administration of anesthesia has not in his opinion reduced the number of lung abscesses. Bevan [88] makes the statement that during a period of two years at the Presbyterian Hospital, Chicago, he has seen from ten to twelve lung and brain abscesses following tonsillectomy. As an argument in favor of the belief that these are hematogenous in origin, he cites several posttonsillectomy brain abscesses occurring without previous lung abscess. The distinction that posttonsillectomy abscess holds in the group of postoperative lung abscesses can probably be partly accounted for by the fact that the operation is conducted in a potentially infected field and the laying open of the venous sinuses is easily accomplished. Likewise the mobility of the area, similarly present in abdominal operations, is conducive to thrombus dislodgment. The influence that mobility exercises toward the breaking loose of a thrombus is strikingly illustrated by the statistics of Cutler and Hunt,[8] in which it was shown that following epigastric operations pulmonary complications developed in 8 per cent of the cases. Grégoire [75] also witnessed such complications in 9 per cent of his cases following gastric surgery under local anesthesia. The time element is of some importance if the complication develops after four to five days. Aspired infections must manifest themselves early. Pulmonary infections developing after a variable symptom free period cannot be regarded in any other way except as embolic in origin. If

89. Hartwell, J. A.: Abscess of the Lung, Ann. Surg. **72**:333 (Sept.) 1920.

pulmonary signs present themselves early, it is not necessary to infer that aspiration is the cause. It is conceivable that emboli can reach the lungs from the operative field as early as the first postoperative day.

Conclusions drawn from the foregoing comment lead us to believe that postoperative lung abscess is in the majority of instances the result of embolism. Could this be proved by experimental investigations, a great weight of evidence would appear in favor of the embolic source of other postoperative pulmonary complications. Such a simplification of the causes of all postoperative pulmonary complications would be entirely reasonable.

II. PREVIOUS ATTEMPTS AT EXPERIMENTAL PRODUCTION

A review of the many varied articles on lung abscess does not disclose a single discourse in which the experimental production of the condition forms the basis for the treatise. Occasional reference, however, reveals that certain procedures have been employed with expectations of a reduplicative lesion, but in all instances the various measures used have been futile. A consideration of the pathogenesis of lung abscess exposes two possible means by which bacteria can be made to reach the desired focus: (1) through the air passages by allowing septic material to be aspirated or by direct transtracheal implantation, and (2) through the blood stream by placing infected material into the venous circulation. The activities of the previous investigators have been entirely centered on the first method of approach, and no attention has been given to the second route of invasion. Aschner [1] was unable to produce the lesion in dogs by means of intratracheal insufflation of pieces of tonsil and adenoid, pus from clinical cases and cultures of anaerobes.

Lambert and Miller [31] were likewise unsuccessful. The uniform presence of anaerobic bacteria in their lung abscess cases led them to believe that such organisms played an important bacteriologic rôle, but when these anaerobes were injected intratracheally into monkeys no lesions developed. Further evidence of the difficulties encountered in producing lung suppuration is demonstrated in a recent article by Scarff.[90] Various bacterial cultures were injected directly into the lung through the chest wall or by opening the chest. Preliminary injections of boiling water and the occlusion of the bronchus with foreign bodies were also carried out as supplementary measures. Failure to produce lung suppuration in any one of the fifty dogs was the result. Although the work of Kline [91] does not concern experimental lung abscess, it is

90. Scarff, J. E.: Pulmonary Blood Pressures: An Experimental Study, Arch. Surg. **12**:591 (Feb.) 1926.

91. Kline, B. S.: Experimental Gangrene, J. Infect. Dis. **32**:481 (June) 1923.

of some significance in that gangrenous pulmonary lesions were developed by the intratracheal injections into rabbits of spirochetes, fusiform bacilli and other organisms obtained from the fluid of a gangrenous lung.

Most investigators contend that lung abscess cannot be produced in animals by intrabronchial manipulations, and deductions based on the meager experimental reports convince us that this is true. In similar experiments carried out in this laboratory the same results have been experienced.

III. PRESENTATION OF EXPERIMENTAL STUDIES AT PRODUCTION

The following experimental studies are based on the two major theories pertaining to the formation of postoperative lung abscess. The material has therefore been classified according to the method used in introducing the infection: (1) by way of the bronchi, and (2) by way of the blood stream. Dogs were used in all the experiments.

INTRABRONCHIAL INJECTION SERIES

Technic.—In a series of fifteen experiments, the bronchoscope was introduced in animals under ether anesthesia following the administration of morphine, one-sixth or one-fourth grain (0.01 or 0.016 Gm.). Various materials, such as freshly obtained bits of human tonsil and adenoid tissue, infected meat and peanut kernels, were then placed by means of a long grasping forceps into the larger and smaller bronchi leading to a single lobe of the lung. At the site of implantation the mucous lining of the bronchus was usually scarified in order that infection might more easily occur. In some animals the lumen of a small bronchus was literally packed with tonsil tissue. In one experiment a small jagged tooth, removed from the animal's jaw, was placed into a lower lobe bronchus. A small chicken feather and a small metallic figure four were likewise used in two other dogs respectively. A mixture of gum tragacanth and cultures of *Bacillus coli* and *Staphylococcus aureus* were also employed. One end of a small, long rubber tube was filled with the mixture; the tube was then introduced through the bronchoscope into a small bronchus and the contents forced into the smaller bronchi with an air pump. In another experiment human tonsil tissue clamped with lead filings was placed into a small bronchus. The animals were carefully watched for the development of a lesion by subsequent bronchoscopic examination and with the aid of roentgenograms.

Results.—The results obtained from this study (table 2) confirm the work of previous investigators in that we were unable to produce a single lung abscess. The materials introduced were usually coughed up during the first few days, although in one instance, namely, the experiment in

which the tooth was used, fourteen days elapsed before expulsion occurred. Three animals succumbed from empyema due to accidental perforation of a bronchus at the time of the initial bronchoscopy. The other animals remained well, and no lesions developed in the region of the implanted foreign body.

TABLE 2.—*Intrabronchial Injection Series*

Experiment	Description of Injection		Resultant Lesion
	Material	Location	
1 (Y 1)	Infected meat.....	Right lower lobe bronchus	Animal died from empyema on third day due to accidental perforation of bronchus at time of instrumentation
2 (Y 2)	Human tonsil.....	Right lower lobe bronchus	None; foreign body expelled
3 (Y 2)	Human tonsil.....	Right lower lobe bronchus	None; foreign body expelled
4 (Y 2)	Human tonsil.....	Right upper lobe bronchus	None; foreign body expelled
5 (Y 2)	Bits of human tonsil.....	Right lower lobe bronchus	No local lesion; animal died from empyema on second day as in experiment 1
6 (Y 3)	Two peanut kernels.....	Right upper lobe bronchus	None; foreign bodies expelled
7 (Y 4)	Human tonsil and adenoid	Right lower lobe bronchus	None; foreign body expelled
8 (Y 4)	Human tonsil.....	Right lower lobe bronchus	None; animal died on first postoperative day from emphyema as in experiment 1
9 (Y 7)	Bits of human tonsil.....	Right lower lobe bronchus	None; foreign body expelled
10 (Y 7)	Bits of human tonsil.....	Right lower lobe bronchus	None; foreign body expelled
11 (Y 12)	Mixture of gum tragacanth and cultures of B. coli and Staphylococcus aureus	Right lower lobe bronchus	None
12 (Y 15)	Mixture of gum tragacanth and cultures of B. coli and Staphylococcus aureus	Right lower lobe bronchus	None
13 (Y 17)	Human tonsil in gum tragacanth and lead filings	Right lower subdivision bronchus	None; foreign body expelled
	Human tonsil and lead filings	Left lower subdivision bronchus	None; foreign body expelled
	Chicken feather.....	Right lower subdivision bronchus	None; foreign body expelled
14 (Y 17)	Small jagged tooth.....	Left lower lobe bronchus	None; foreign body expelled after two weeks
	Mixture of gum tragacanth and cultures of B. coli and Staphylococcus aureus	Right lower lobe bronchus	None
15 (Y 17)	Small metalic figure 4.....	Left lower lobe bronchus	None; foreign body expelled

PROTOCOLS

EXPERIMENT 1.— Dog Y1, weighing 8 Kg., Feb. 19, 1925, was given one-sixth grain (0.01 Gm.) morphine and $\frac{1}{150}$ grain atropine. Under ether anesthesia the bronchoscope was introduced into the right primary bronchus and then into the bronchus leading to the lower lobe. A long wire hook was introduced and the wall of the bronchus was scarified. A small bit of cooked meat, which had been rubbed on the floor, was then introduced with a hook forceps and pushed through the wall of the bronchus presumably into the lung tissue itself.

February 21, the dog appeared ill and the respiratory rate was increased.

February 22, the dog was found dead in his cage. Necropsy revealed a bilateral empyema. A small perforation led from the bronchus into the pleural cavity. The pleural infection evidently resulted from perforation into the pleural cavity at the time of instrumentation.

EXPERIMENT 2.—Dog Y2, weighing 5.7 Kg., March 5, 1925, was given one-sixth grain (0.01 Gm.) of morphine and 1/150 grain of atropine. Under ether anesthesia a 9 mm. bronchoscope was introduced into the right lower lobe bronchus. The wall of the bronchus was scarified with a long wire hook and a section of human tonsil was implanted next to the injured bronchial wall.

March 19, the dog was submitted to bronchoscopic examination under ether anestheia. The foreign body introduced two weeks previously could not be found and no signs of inflammation were present. The same area on the bronchial wall was scarified.

April 2, a preliminary hypodermic injection of one-sixth grain of morphine and 1/100 grain of atropine was given followed by ether anesthesia. Bronchoscopic examination revealed a normal bronchus.

EXPERIMENT 3.—Dog Y2, April 2, 1925, was given one-sixth grain of morphine and 1/100 grain of atropine. Under ether anesthesia the bronchoscope was introduced into the right lower lobe bronchus. The bronchial wall was again scarified until bleeding occurred and a small piece of freshly obtained human tonsil containing caseous cryptic material was placed next to the injured bronchial wall. Cultures for the predominating organisms were taken from the tonsil.

April 6, bronchoscopic examination revealed no evidence of inflammation. The foreign body could not be found. The bacteria found in the culture consisted of short chain streptococci, staphylococci and gram-negative bacilli.

EXPERIMENT 4.—Dog. Y2, April 16, 1925, was given one-sixth grain of morphine and 1/100 grain of atropine. The bronchoscope was introduced with the dog under ether anesthesia. The right upper bronchus was scarified until bleeding occurred. A large piece of human tonsil sufficient in size to occlude the bronchus was introduced.

April 30, bronchoscopic examination revealed the bronchial lumen to be patent. There were no signs of inflammation.

EXPERIMENT 5.—Dog Y2, April 30, 1925, was given one-sixth grain of morphine and 1/100 grain of atropine. Under ether anesthesia, the bronchoscope was introduced and finely chopped bits of human tonsil tissue were placed into the right lower bronchus blocking it entirely.

May 1, the dog appeared sick and would not eat.

May 2, the dog was found dead in his cage. Necropsy showed a bilateral empyema. A small perforation led from a lower lobe bronchus into the pleural cavity. The pleural infection was attributed to perforation of the bronchoscope into the pleural cavity.

EXPERIMENT 6.—Dog Y3, weighing 9.4 Kg., May 28, 1925, was given one-sixth grain of morphine and 1/100 grain of atropine. Under ether anesthesia the bronchoscope was introduced into the right upper main stem bronchus and two pieces of peanut, constituting approximately one-half the kernel, were inserted into the bronchial lumen.

June 8, bronchoscopic examination showed no inflammation. The foreign bodies were missing.

June 17, the dog was killed in a fight. There was no evidence of an infectious process.

EXPERIMENT 7.—Dog Y4, weighing 14.6 Kg., May 28, 1925, was given one-fourth grain of morphine and 1/100 grain of atropine. Under ether anesthesia the bronchoscope was introduced into the right lower lobe bronchus and the lumen was entirely filled with human tonsil and adenoid tissue.

June 2, bronchoscopic examination revealed no inflammation of the bronchial mucous membrane. The tonsil tissue had been expelled.

EXPERIMENT 8.—Dog Y4, July 2, 1925, was placed under ether anesthesia and the bronchoscope introduced into the right lower lobe bronchus. An entire tonsil was cut into small pieces and inserted into the bronchial lumen.

July 3, the dog was found dead in his cage. Immediately after the introduction of the instrument the dog developed respiratory distress and cyanosis which grew progressively worse until death. At necropsy a perforation was found in the lung leading to a right lower lobe bronchus. The tonsil was found remaining in the bronchial lumen, but there was no evidence of inflammation. The cause of death was attributed to a pneumothorax.

EXPERIMENT 9.—Dog Y7, weighing 9.4 Kg., July 25, 1925, was given one-sixth grain of morphine and 1/150 grain of atropine. Under ether anesthesia the bronchoscope was introduced and three bits of human tonsil tissue were placed into the right lower lobe bronchus.

July 28, bronchoscopic examination revealed an absence of the tonsil tissue and no inflammatory change was visible.

EXPERIMENT 10.—Dog Y7, Aug. 7, 1925, was given one-sixth grain of morphine and 1/150 grain of atropine. Under ether anesthesia the bronchoscope was introduced and the right lower lobe bronchus filled with human tonsil tissue.

August 27, the dog was found dead in the kennels. Death was due to trauma received in a fight. The right lower bronchus gave evidence of a slight purulent inflammation also present throughout the bronchial tree. There were a few small peribronchial patches of infiltration throughout. The animal probably had distemper.

EXPERIMENT 11.—Dog Y12, weighing 10.5 Kg., Oct. 2, 1925, was given one-fourth grain of morphine. Under ether anesthesia, the bronchoscope was introduced and a mixture of gum tragacanth, *Bacillus coli* and *Staphylococcus aureus* forced through a rubber tube into the terminal bronchi of the right lower lobe by means of an air pump. The compressed chest was released at the same time so as to create a forceful inspiration.

October 5, roentgenograms of the chest showed no evidence of lung involvement.

October 8, roentgenograms of the chest revealed no altered appearance of the lungs.

October 14, roentgenograms revealed clear lung fields. Bronchoscopic examination showed no evidence of bronchial inflammation. There seemed to be an odor of *Bacillus coli* to the breath.

EXPERIMENT 12.—Dog Y15, weighing 6.4 Kg., Oct. 3, 1925, was given one-sixth grain of morphine. Under ether anesthesia the bronchoscope was introduced and a mixture of gum tragacanth, *Bacillus coli* and *Staphylococcus aureus* forced through a rubber tube into the terminal bronchi of the right lower lobe by means of an air pump. The compressed chest was released at the same time so as to create a forceful inspiration.

October 8, roentgenograms of the chest showed the lung fields to be clear.

October 14, roentgenograms of the chest revealed no change in density of the right lower lobe. Bronchoscopic examination showed no evidence of inflammation in the bronchi although here again the odor of *Bacillus coli* was detected.

EXPERIMENT 13.—Dog Y17, weighing 6.7 Kg., Sept. 21, 1925, was given one-sixth grain of morphine. Under ether anesthesia the bronchoscope was introduced and a piece of human tonsil rolled in gum tragancanth and fastened with lead filings was placed into a right lower subdivision bronchus. A piece of human tonsil clamped with lead was placed into a left lower subdivision bronchus and a small chicken feather was also placed into another one of the right lower bronchi.

September 22, a roentgenogram of the chest showed the one foreign body still present in the right lower lobe. The one on the left had disappeared. There was no evidence of any pathologic change.

September 26, a roentgenogram of the chest revealed the absence of both foreign bodies.

October 2, bronchoscopic examination revealed no evidence of inflammation. The foreign bodies had disappeared.

EXPERIMENT 14.—Dog Y17, Oct. 2, 1925, was given one-sixth grain of morphine. Under ether anesthesia, the bronchoscope was introduced and a jagged tooth of the animal was placed into the left lower lobe bronchus. A mixture of gum tragacanth, *Bacillus coli* and *Staphylococcus aureus* was also forced into the terminal bronchi of the right lower lobe in the same manner as in experiment 11.

October 5, roentgenograms of the chest revealed a faint shadow in the region of the implanted tooth, probably the foreign body itself. No evidence of a pathologic change was discernible.

October 8, roentgenograms of the chest revealed clear pulmonary areas.

October 14, roentgenograms of the chest showed no change from the preceding plates. Bronchoscopic examination revealed no evidence of inflammation in the bronchi but here again the odor of *Bacillus coli* was detected. The tooth was still present in the left lower lobe bronchus but no pus or redness of the bronchus was visible.

October 23, bronchoscopic examination showed the tooth to be absent. There was again no inflammation present.

EXPERIMENT 15.—Dog Y17, Oct. 23, 1925, was given one-sixth grain of morphine. Under ether anesthesia the bronchoscope was introduced and a metallic figure 4 was inserted into the same left lower lobe bronchus that the tooth had occupied.

October 29, roentgenograms of the chest revealed the foreign body still present in the left lower lobe. No infiltration could be seen about the foreign body.

November 15, the dog died of distemper. Examination of the left lung failed to locate the foreign body. The bronchus that contained the metallic figure 4 was of normal size. The entire bronchial tree showed the diffuse characteristic appearance of distemper.

INTRAVENOUS INJECTION SERIES (TABLE 3)

Technic.—Following and in conjunction with the discouraging results experienced with the intrabronchial methods, the idea was conceived of introducing into the large veins infected materials, which would be transported to the right side of the heart by way of the venous circulation and thence to the lung by way of the pulmonary artery. The ease with which foreign bodies can be placed within the jugular vein because of its size and accessibility and the absolute certainty of subsequent transmission to the right side of the heart led us to choose this vessel as the most suitable receptacle for such materials.

TABLE 3.—*Intravenous Injection Series*

Experiment	Description of Embolus			Roentgen-Ray Diagnosis of Lesion	Duration of Lesion, Days	Pathologic Description of Specimen	
	Material	Organisms	Location			Gross	Microscopic
16 (Y 5)	Tonsil.....	.....	Right lower lobe	Abscess	5	Large necrotic walled abscess perforating into bronchus and pleural cavity	Necrotic abscess wall with an adjoining zone of acute diffuse inflammation and densely infiltrated with leukocytes
17 (Y 6)	Gelatin capsule containing tonsil and lead	.....	Left lower lobe	Abscess	8	Few pleural adhesions; three small abscesses in lobe	Beginning resolution; moderate diffuse acute inflammation
18 (Y 8)	Gelatin capsule containing tonsil and lead	.....	Left lower lobe	Abscess	33	Large area of fibrosis in lobe surrounding lead filing	
19 (Y 9)	Gelatin capsule containing tonsil and lead	.....	Left lower lobe	None	..		
20 (Y 9)	Fat, lead.....	Staphylococcus aureus, B. coli	Left lower lobe	Abscess	14	Small fibrotic walled abscess.....	Fibrosis of abscess wall; considerable round cell infiltration still present
21 (Y 10)	Muscle, lead....	Staphylococcus aureus	Left lower lobe	Abscess	13	Small abscess in tip of lobe.....	Resolution well established; fibrosis of abscess wall
22 (Y 11)	Muscle, metal tube	Staphylococcus aureus	Left lower lobe	Infiltration	19	Normal	
23 (Y 12)	Potato.....	Staphylococcus aureus	.....	None	..		
24 (Y 13)	Potato, lead shot, muscle	Staphylococcus aureus	Left lower lobe	Abscess	14	Two small abscesses containing shot and potato	Beginning fibrous tissue formation of abscess walls; considerable cellular exudate still present
25 (Y 14)	Muscle, lead....	Staphylococcus aureus	Left lower lobe	Infiltration	14	Surface scarring of lobe with an underlying fibrotic infarct	Resolution beginning; still moderate infiltration
26 (Y 15)	Muscle, lead....	Staphylococcus aureus	Left lower lobe	None	..		
27 (Y 16)	Tonsil lead.....	Staphylococcus aureus (supplementary)	Left lower lobe	Abscess	8	Lobe consolidated and containing large abscess perforating into pleural cavity	Necrotic abscess wall with an adjoining zone of acute diffuse inflammation
28 (Y 12)	Vein, lead, blood	Staphylococcus aureus	Right lower lobe	None	..		
29 (Y 18)	Vein, lead, blood	Staphylococcus aureus, B. coli	Left lower lobe	Abscess	10	Small abscess in lobe.....	Fibrosis of abscess wall; inflammation resolving
30 (Y 19)	Vein, lead, blood	Staphylococcus aureus, B. coli	Left lower lobe	Abscess	34	Small fibrotic walled abscess.....	Still considerable small cell infiltration in wall; fibrous tissue lining
31 (Y 20)	Vein, lead, blood	Staphylococcus aureus, B. coli	Left lower lobe	Abscess	6	Large necrotic wall abscess.....	Inner wall necrotic; surrounding lung tissue densely infiltrated with leukocytes
32 (Y 21)	Vein, lead, blood	B. coli.....	Left lower lobe	Abscess	33	Healed area of fibrosis	
33 (Y 22)	Vein, blood, lead	B. coli.....	Left lower lobe	Infiltration	15	Area of fibrosis about lead.....	Almost complete fibrotic organization
34 (Y 23)	Vein, blood, lead	Staphylococcus aureus, B. coli, streptococcus	Right upper lobe	Consolidation	58	Area of fibrosis about lead	
35 (Y 24)	Vein, blood, lead	Staphylococcus aureus, B. coli, streptococcus	Right lower lobe	Abscess	33	Small cavity almost obliterated with fibrosis	Complete fibrotic healing
36 (Y 25)	Vein, blood, lead	Pneumococcus, streptococcus	Left lower lobe	Infiltration	46	Area of fibrosis about lead	
37 (Y 26)	Vein, blood, lead	Pneumococcus, streptococcus	Left lower lobe	Consolidation	..		

38 (Y 27)	Vein, blood, lead	Staphylococcus aureus	Right lower lobe	Abscess	27	Small abscess cavity in lobe.....	Smooth walled zone of fibrous tissue; almost completely healed
39 (Y 28)	Vein, blood, lead	Staphylococcus aureus	Left lower lobe	None	..	Normal	
40 (Y 29)	Vein, blood, lead	Streptococcus	Left lower lobe	Abscess	23	Small abscess cavity in lobe.....	Fibrosis of inner wall; process almost healed
41 (Y 30)	Vein, blood, lead	Streptococcus	Right lower lobe	Abscess	16	Small abscess cavity.....	Smoothly lined fibrotic cavity; small amount of cellular infiltration
42 (Y 31)	Vein, blood, lead	Streptococcus	Left lower lobe	Abscess	23	Small abscess cavity.....	Fibrous wall only remained; inflammation resolved
43 (Y 32)	Vein, blood, lead	Pneumococcus type II	Right lower lobe	None	15	Area of fibrosis about lead	
44 (Y 33)	Vein, blood, lead	Pneumococcus type II	Right lower lobe	None	19	Area of fibrosis about lead.....	Fibrous tissue formation; few round cells present
45 (Y 34)	Vein, blood, lead	Pneumococcus type II	Right lower lobe	None	14	Area of fibrosis about lead	
46 (Y 35)	Vein, blood, lead	Staphylococcus aureus, pneumococcus type II	Right lower lobe	Abscess	22	Multiloculated abscess cavity 2.5 cm. in diameter	Smooth walled fibrotic cavity; considerable round cell infiltration
47 (Y 36)	Vein, blood, lead	Staphylococcus aureus, pneumococcus type II	Right lower lobe	Abscess	21	Large abscess in lobe.....	Fibrotic walled cavity; moderate degree of cellular infiltration
48 (Y 37)	Vein, blood, lead	Staphylococcus aureus, pneumococcus type II	Right lower lobe	Infiltration	17	Area of fibrosis about lead	
49 (Y 38)	Vein, blood, lead	Staphylococcus aureus, B. coli, pneumococcus	Left lower lobe	Abscess	16	Small abscess in lobe.....	Moderate degree of cell exudation in wall; some fibrosis
50 (Y 39)	Vein, blood, lead	Staphylococcus aureus, B. coli, pneumococcus type II	Right lower lobe	Abscess	12	Large abscess in lobe.....	Inner wall of abscess necrotic; moderate degree of diffuse inflammation
51 (Y 40)	Vein, blood, lead	Staphylococcus aureus, B. coli, pneumococcus type II	Right lower lobe	Abscess	13	Area of fibrosis about lead.....	Almost healed; dense fibrous tissue formation
52 (Y 48)	Vein, blood, lead	Staphylococcus aureus, B. coli, pneumococcus and streptococcus	Right lower lobe	Abscess	..		
53 (Y 51)	Vein, blood, lead	Staphylococcus aureus, B. coli, pneumococcus and streptococcus	Left lower lobe	Abscess	12	Lung collapsed; small abscess perforating into pleural cavity	
54 (Y 52)	Vein, blood, lead	Staphylococcus aureus, B. coli, pneumococcus and streptococcus	Left lower lobe	Abscess	8	Small abscess in lobe	
55 (Y 59)	Vein, blood, lead	B. coli and streptococcus	Left lower lobe	Abscess	4	Large necrotic walled abscess	
56 (Y 60)	Vein, blood; lead	B. coli and streptococcus	Right middle lobe	Abscess	10	Small abscess in lobe	

Method of Introducing Embolus: The animal is anesthetized with ether, following the administration of ⅙ or ¼ grain of morphine hypodermically. The right jugular vein is exposed and isolated for about 3 cm. Single strands of silk loops weighted with clamps, or bulldog clamps, are placed about the central and distal portions of the exposed vessel to control bleeding. A transverse incision is then made to receive a glass cannula, sufficiently large in diameter to fill the vein lumen. The cannula is connected with rubber tubing to a Luer syringe, and the entire system is filled with salt solution. The material to be injected is then placed into the cannula; the cannula is inserted into the vein; the proximal bleeding control is released, and the embolus is shot into the circulation. To insure passage of the embolus downward the syringe can be filled and emptied several times. The opening in the vein is sutured to reestablish the lumen, or the vessel is merely ligated above and below.

Following the intravenous injection, daily roentgenograms of the chest are taken, and the development of a pulmonary lesion carefully watched for.

Results.—In the first few experiments (experiments 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11 and 12) various materials were used as emboli, such as pieces of infected tonsillar tissue, bits of muscle and fat impregnated with different bacteria, and in two instances a small cylinder of potato injected with an emulsion of *Staphylococcus aureus*. If such substances, however, are permitted to circulate freely in the blood stream, the bacteria implanted in the bits of tissue can easily become detached and a distribution over a large pulmonary area instead of a concentration at one point will result. In order to prevent the bacteria from being scattered, a small sterile gelatin capsule was used as the conveyor; the infected material was placed into the capsule and the filled capsule injected. The rate of absorption of the gelatin is such that the capsule will not dissolve until lodgment in a pulmonary vessel has taken place. A small bit of paraffinized lead was also included in order that the final resting place of the embolus could be determined by roentgenograms. It was thought that any possible deleterious effect of the metal on the bacteria could be averted if paraffin was used as a coating.

Difficulty was experienced, however, in obtaining gelatin capsules small enough to allow free venous introduction necessary for further promulgation. Their use was therefore abandoned and pieces of muscle and fat tissue were merely folded over a culture of bacteria and the tissue clipped together with a lead filing. In one instance a hole was bored into a small lead shot, bacteria were placed into the hole, and the opening was closed with a bit of muscle tissue. In still another experiment a small metal tube was filled with a bacterial culture, and both ends were closed with muscle.

The placement of infected materials into the venous circulation in these preliminary experiments demonstrated the comparative ease with which lung abscess could be produced, and the information obtained furnished sufficient evidence to justify further efforts toward constructing more uniform emboli.

The method finally used in preparing the artificial, infected embolus is as follows: The femoral vein is exposed, ligated centrally and distally, and a portion, approximately from 6 to 8 mm. long, utilized for the embolus. A longer strip of vein can be removed so that two or three emboli can be made from the same segment of vein. One end of a

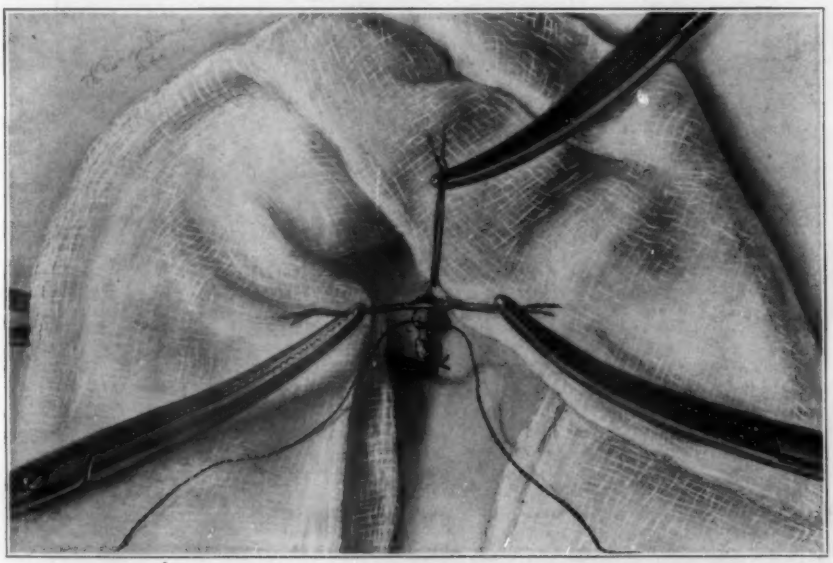

Fig. 1.—Method of preparing vein embolus: One end of the vein segment is closed with a silk ligature; a straight clamp holds this end down firmly; three guy sutures of silk attached to curved clamps serve to hold open the other end of the vein segment; a small bit of lead, bacteria and a few drops of blood can then be introduced; the open end is closed with a ligature of silk.

6 to 8 mm. segment is ligated with silk and held stationary by an attached straight clamp. The other end is held open by three silk sutures as illustrated in figure 1. Into the lumen of this small segment of vein an emulsion of bacteria is introduced by a platinum loop, together with a small bit of lead, previously coated with paraffin so as to render it inert. A few drops of blood are then added with a blunt pointed needle attached to a syringe, the point of the needle being tied in with the silk ligature about the upper end of the vein segment. This is to prevent spilling of infected blood and to enable the production of

slight pressure so that the embolus can be brought to the desired 3 to 4 mm. diameter. A tight grasp on the ligature is maintained while the needle is withdrawn so that when the end is ligated, bacterial contamination of the exterior is reduced to a minimum. The embolus, resembling a small sausage, is now placed into the open end of the glass cannula and injected (fig. 2).

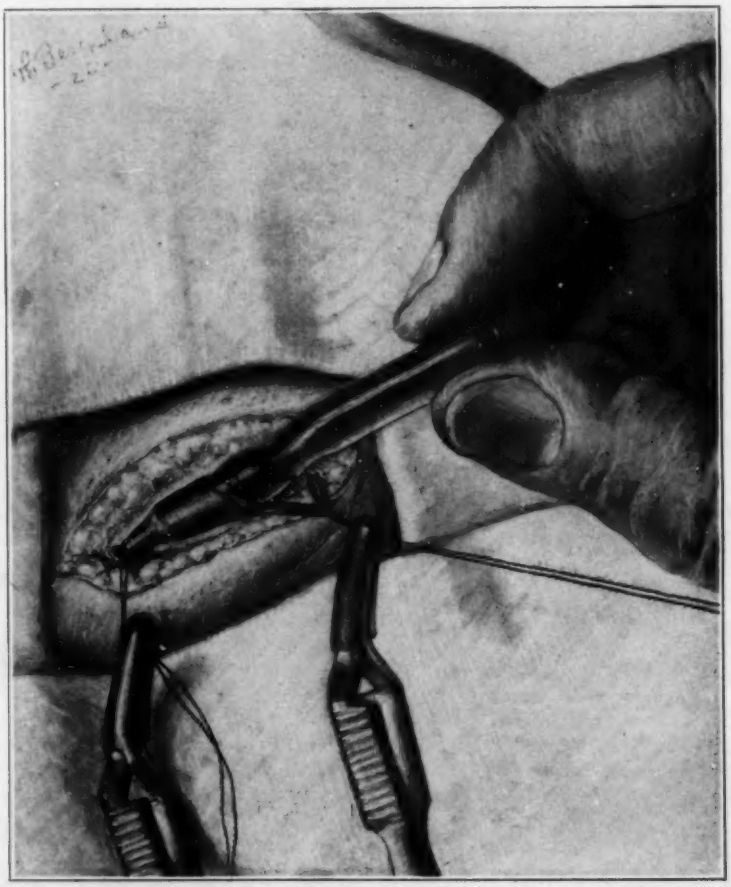

Fig. 2.—Method of introducing septic embolus into jugular vein: A glass cannula with an attached rubber tubing and syringe is filled with salt solution; the embolus is placed into the open end of the cannula, the cannula is inserted into the vein lumen and the embolus is washed into the circulation.

The development of a standard method for the construction and injection of the embolus suggested the possibility of selecting the organism most concerned in abscess formation. The following bacteria were used: *Staphylococcus aureus,* nonhemolytic streptococcus, *Bacillus*

TABLE 4.—*Incidence of Abscess with Various Organisms*

Organism	Experiment	Resultant Lesion
Staphylococcus aureus	21 (Y 10)	Abscess formation on sixth day; still present at necropsy on thirteenth day
	22 (Y 11)	Infiltration on sixth day with subsidence on eighteenth day
	23 (Y 12)	None
	24 (Y 12)	None
	25 (Y 13)	Abscess formation on seventh day; cavity still present at lobectomy on fourteenth day
	26 (Y 14)	Infiltration beginning on third day with subsidence on eleventh day
	27 (Y 15)	None
	38 (Y 27)	Abscess formation on fifth day; large cavity still present at lobectomy on twenty-fifth day
	39 (Y 28)	None
Streptococcus.....	40 (Y 29)	Abscess formation on fifth day; large cavity still present at lobectomy on twenty-third day
	41 (Y 30)	None
	42 (Y 31)	Abscess formation on tenth day; large cavity still present at lobectomy on twenty-third day
Bacillus coli.....	32 (Y 21)	Abscess formation on fifth day; lesion healed on thirty-first day
	33 (Y 22)	Infiltration beginning on fifth day with subsidence on ninth day
Pneumococcus.....	43 (Y 32)	None
	44 (Y 33)	None
	45 (Y 34)	None
Staphylococcus aureus, B. coli	20 (Y 9)	Abscess formation on fifth day; small fibrotic walled abscess still present at lobectomy on fourteenth day
	29 (Y 18)	Abscess formation beginning on eighth day; small cavity found at lobectomy on tenth day
	30 (Y 19)	Abscess formation on sixth day; small cavity still present at lobectomy on thirty-fourth day
	31 (Y 20)	Abscess formation on fourth day; large cavity still present at lobectomy on sixth day
Staphylococcus aureus, pneumococcus	46 (Y 35)	Abscess formation on fourth day; large cavity present at necropsy on twenty-second day
	47 (Y 36)	Abscess formation on sixth day; large cavity present at lobectomy on twenty-first day
	48 (Y 37)	Infiltration beginning on seventh day; lesion found healed at lobectomy on nineteenth day
Staphylococcus aureus, B. coli pneumococcus	49 (Y 38)	Abscess formation on fifth day; large abscess still present at lobectomy on sixteenth day
	50 (Y 39)	Abscess formation beginning on eighth day; large cavity found at lobectomy on twelfth day
	51 (Y 40)	Abscess formation on fifth day; fibrotic lesion found at lobectomy on thirteenth day
Streptococcus, pneumococcus	36 (Y 25)	Slight infiltration beginning on sixth day with subsidence after eight days
	37 (Y 26)	Beginning consolidation left lower lobe on first postoperative day and abscess formation on third day; resolution on eighteenth day
Staphylococcus aureus, streptococcus, B. coli	34 (Y 23)	Consolidation right upper lobe on fifth day with resolution on twenty-fifth day
	35 (Y 24)	Abscess formation on fifth day; small fibrotic walled cavity still present at time of lobectomy on thirty-third day
Staphylococcus aureus, streptococcus, pneumococcus, B. coli	52 (Y 48)	Abscess formation on seventh day; resolution complete on fourteenth day
	53 (Y 51)	Abscess formation on seventh day with death from rupture into pleural cavity on eleventh day
	54 (Y 52)	Abscess formation on fifth day; death from rupture into pleural cavity on eighth day
B. coli, streptococcus	55 (Y 59)	Abscess formation on second day; large necrotic walled abscess present at lobectomy on fourth day
	56 (Y 60)	Abscess formation on sixth day; small abscess present at lobectomy on tenth day

coli and pneumococcus (table 4). No attempt was made to use bacteria of the same strain and virulence as interest lay chiefly in obtaining a single organism or group of organisms which would invariably cause an abscess. *Staphylococcus aureus* was thought to be most suitable because of certain attributes possessed by this organism, in particular the liquefying action on tissues. In the nine experiments in which *Staphylococcus aureus* was employed as the sole organism, pulmonary lesions failed to develop in four animals. In two other animals infiltration occurred and in only three animals did a true abscess develop. This seemed to indicate that the dog maintains a fairly high resistance to the staphylococcus. The colon bacillus is likewise a tissue liquefier, and in the two experiments in which this organism was used alone an infiltrative lesion developed in one instance without abscess formation, while in the second experiment a lung abscess followed. When these two organisms, however, were combined, lung abscess developed in each of the four animals used. The nonhemolytic streptococcus was responsible in causing two lung abscesses. In a third animal no lesion developed. A pneumococcus was used in three animals without the development of a single lesion. This particular organism had been freshly obtained from a mouse previously inoculated with the sputum from a pneumonia patient. This same organism, however, when combined with *Staphylococcus aureus* and enclosed in the embolus produced an abscess in two out of three animals.

It is evident, therefore, that in the dog a lessened susceptibility exists to any of the foregoing organisms when used singly, and that this is especially true in the case of the pneumococcus and to a lesser extent in the case of the staphylococcus. The colon bacillus and the streptococcus are more apt to produce an infection when used singly. When, however, certain combinations of the organisms are employed, such as a mixture of *Staphylococcus aureus* and *Bacillus coli,* lung abscess will usually result, and if in addition streptococci and pneumococci are introduced into the embolus, the experiments seem to indicate that lung abscess invariably results.

In order to show that emboli may reach the lung from any operative field where the venous channel is unimpeded by valves, we performed two experiments (experiment 55, Dog Y59, and experiment 56, Dog Y60) in which a vein embolus inoculated with cultures of *Bacillus coli* and streptococcus was inserted into the femoral vein. A segment was excised from the distal and smaller portion of the femoral vein; the segment was prepared in the usual manner; the cannula containing the embolus was inserted into the lumen of the central portion of the vein, and the infected embolus was washed into the circulation with salt solution. A lung abscess developed in both animals.

PROTOCOLS

EXPERIMENT 16.—Dog Y5, weighing 6.5 Kg., July 18, 1925, was given one-fourth grain of morphine. Under ether anesthesia, the right jugular vein was exposed. Bulldog clamps were placed on the vein centrally and distally and the vessel was opened by a transverse incision. A small piece of tonsil was inserted into the vein and the tissue forced downward with salt solution. The opening in the vein was sutured with silk and the skin was closed with a continuous subcuticular silk suture.

July 21, the dog appeared sick and would not eat.

July 22, the dog continued ill. The respiratory rate was definitely increased. A roentgenogram of the chest showed considerable infiltration of the right lower lobe.

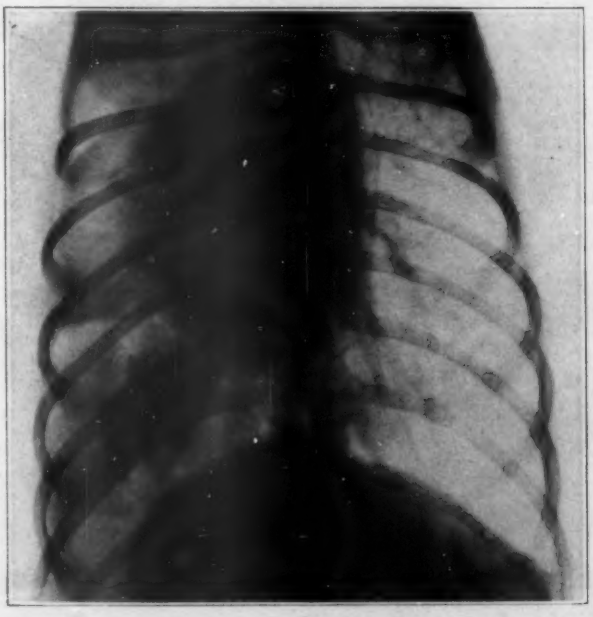

Fig. 3.—Dog Y5, experiment 16, five days after embolism; the upper area of the consolidated right lower lobe is less dense and probably indicates the upper limits of the abscess.

July 23, there was a marked degree of dyspnea. Just preceding death a bloody discharge was seen to issue from the nostrils. A roentgenogram of the chest taken before death revealed a consolidated lower right lobe containing areas of lessened density (fig. 3).

Necropsy.—The pleural cavity contained a cloudy hemorrhagic fluid. The left lung was normal. The right lower lobe was greatly increased in size, red and firm, and presented a fibrinous exudate on the pleural surface. A necrotic perforation entered what was found to be on section a large irregular necrotic walled abscess cavity filled with a partially organized blood clot (fig. 4). An opening led into a bronchus and blood was seen throughout the entire bronchial tree.

Microscopically, the abscess wall consisted of necrotic tissue with an adjoining zone of acute diffuse inflammation.

EXPERIMENT 17.—Dog Y6, weighing 18.8 Kg., July 24, 1925, was given one-fourth grain of morphine. Under ether anesthesia, the right jugular vein was exposed and opened transversely. Bleeding was controlled by single silk strands weighted with clamps. A gelatin capsule filled with tonsil segments and a piece of paraffin coated lead filing was placed into the vessel and forced downward with salt solution. The opening in the vein was closed with silk and the skin was approximated with a continuous subcuticular stitch of silk. A roentgenogram of the chest taken after the operation localized the foreign body in the left lower lobe.

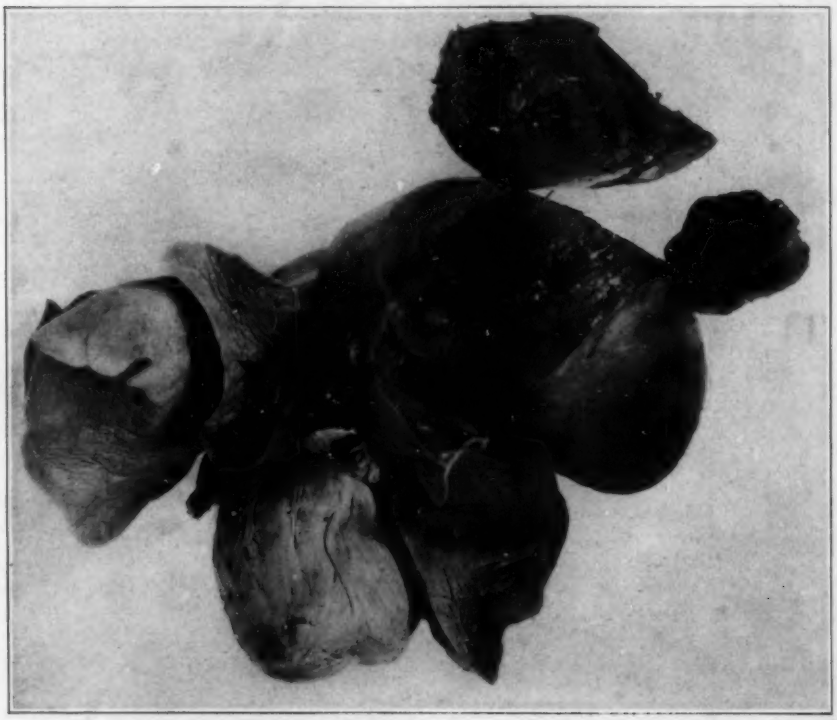

Fig. 4.—Lung specimen obtained at necropsy from Dog Y5, experiment 16, five days after embolism; the right lower lobe contains a large abscess.

July 29, a roentgenogram of the chest showed a large area of density with a well defined central clear area present in the left lower lobe. The respiratory rate was definitely increased.

August 1, the dog was killed in a fight.

Necropsy.—There were some recent pleural adhesions existing between the left lower lobe and the chest wall. The left lower lobe was increased in size and density. The surface presented a reddened area at one point, and section through this area revealed one fairly large abscess. Two small abscesses adjoined the larger one (fig. 5). The walls of the abscesses were roughened and necrotic. Microscopically, the abscess wall showed a rather acute diffuse inflammation of the lung tissue adjoining an area of necrosis. Some fibrous tissue was present, a beginning resolution.

EXPERIMENT 18.—Dog Y8, weighing 18 Kg., Aug. 7, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was exposed. Bleeding was controlled in the routine manner; the vein was opened by a transverse incision; a gelatin capsule filled with a paraffinized lead filing and a small piece of human tonsil was inserted into the lumen of the vessel; the capsule was forced on with salt solution and the opening in the vessel was closed with silk. The skin was approximated with the usual subcuticular stitch of silk. A roentgenogram localized the foreign body in the left lower lobe.

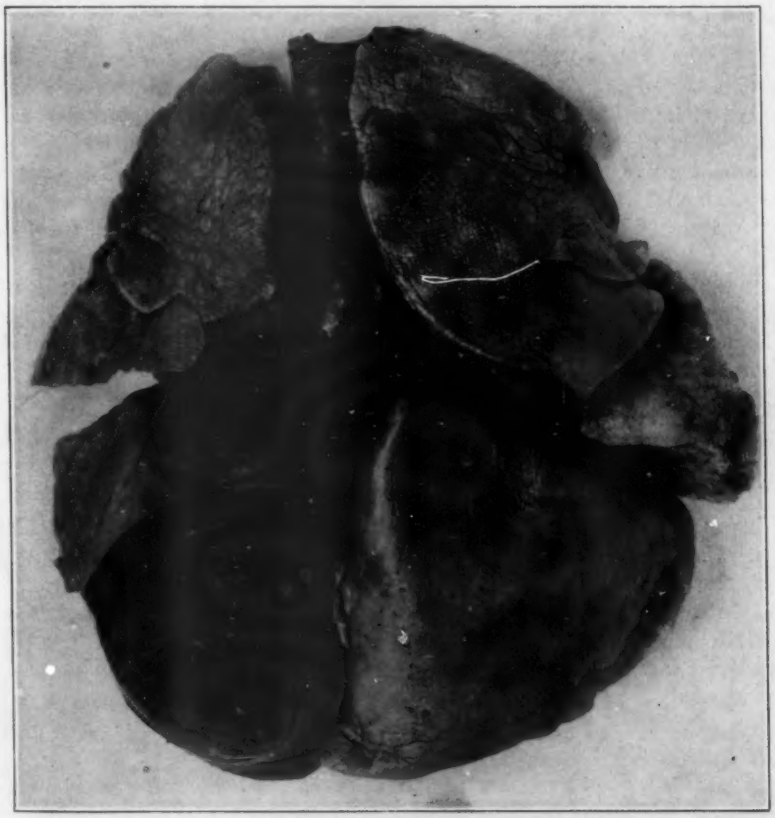

Fig. 5.—Lung specimen obtained at necropsy from Dog Y6, experiment 17, eight days after embolism; the left lower lobe contains one large and two small abscesses.

August 11, a roentgenogram of the chest showed a diffuse cloudiness of the left lower lobe with a suggestive area of cavitation about the piece of lead. The dog did not eat his food well.

August 20, a roentgenogram of the chest disclosed an abscess approximately 2 cm. in diameter in the left lower lobe just above the dome of the diaphragm near the hilum. The dog appeared well.

August 26, the dog had apparently overcome his infection. A roentgenogram of the chest still showed a small cavity. The dog was removed to the main kennels.

September 3, a roentgenogram of the chest showed complete obliteration of the cavity.

September 9, the dog was killed in a fight.

Necropsy.—There were numerous wounds covering the entire body. The left pleural cavity contained numerous adhesions binding the left lower lobe to the diaphragm and to the lateral chest wall. There was considerable puckering at the lower pole of the lobe and a thickened area about the size of a walnut could be felt within the lobe. The lung cut with resistance at this point. The lead filing was recovered completely encased in fibrous tissue. Sections for microscopic examination were not taken.

EXPERIMENT 19.—Dog Y9, weighing 12 Kg., Sept. 10, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was exposed and prepared for reception of the embolus in the routine manner. A gelatin capsule loaded with a piece of human tonsil tissue and a paraffinized piece of lead filing was introduced into the lumen of the vein and forced onward with salt solution. The vein was closed with silk and the skin was approximated with a subcuticular stitch of silk. A roentgenogram of the chest revealed the foreign body in the left lower lobe.

September 12, the dog was apparently well. No change from the previous roentgenogram was noted.

September 15, a roentgenogram of the chest disclosed no evidence of lung inflammation.

September 21, there was no evidence of infiltration as shown by the roentgenogram.

September 30, repeated roentgenograms of the chest revealed no evidence of a lung lesion.

EXPERIMENT 20.—Dog Y9, Sept. 30, 1925, was given one-fourth grain of morphine. Under ether anesthesia the left jugular vein was exposed and prepared for the reception of an embolus in the routine manner. A piece of fat removed from the wound was folded over a culture of *Staphylococcus aureus* and *Bacillus coli* and clipped together with a paraffinized lead filing. The material was placed into a glass cannula, the cannula was inserted into the lumen of the vein and the embolus was forced onward with salt solution. The vein was ligated above and below with silk, and the skin was closed with subcuticular silk. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 2, a roentgenogram of the chest revealed a circumscribed area of beginning density about the lead filing.

October 5, a roentgenogram of the chest showed a greater degree of infiltration with beginning rarefaction immediately surrounding the piece of lead.

October 7, cavity formation about the lead was quite distinct

October 12, cavity formation was marked, but the infiltration in the surrounding lung tissue was of less extent.

October 14, it was decided to remove the lobe and a lobectomy was performed. The specimen showed no surface scarring. There was some increase in size and density. When the lung was sectioned, a small abscess was encountered with some apparent fibrosis of the walls (fig. 6). Microscopically, a section of the abscess wall showed considerable fibrosis. Considerable cellular infiltration, chiefly round cells, was seen in the surrounding lung tissue. Histologically resolution was established.

EXPERIMENT 21.—Dog Y10, weighing 6.8 Kg., Sept. 11, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was exposed and prepared for reception of the embolus in the routine manner. A piece of muscle taken from the wound was folded about a culture of *Staphylococcus aureus* and clamped with a piece of paraffinized lead filing. The material

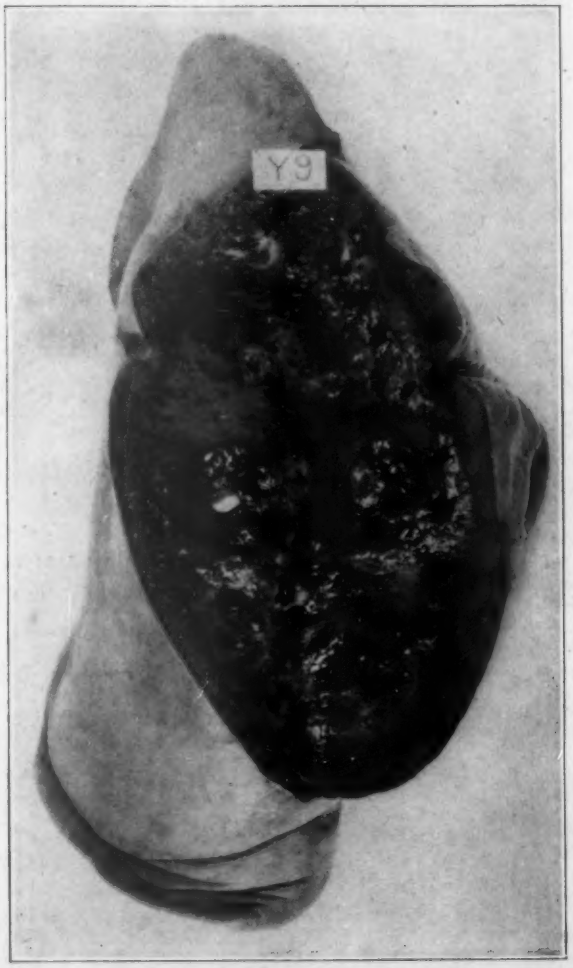

Fig. 6.—Left lower lobe removed from Dog Y9, experiment 20, fourteen days after embolism; a small fibrotic walled abscess still remains.

was then placed into a glass cannula, the cannula inserted into the vein and the embolus forced onward with salt solution. The vein was ligated and the skin approximated with a subcuticular stitch of silk. A roentgenogram of the chest localized the foreign body in the left lower lobe.

September 14, a roentgenogram of the chest showed some density about the foreign body.

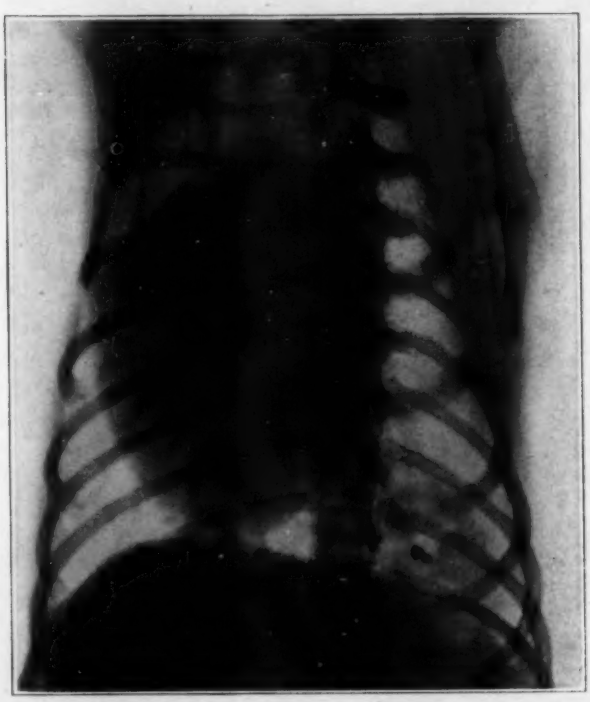

Fig. 7.—Dog Y10, experiment 21, seven days after embolism; the lead filings can be seen lying at the bottom of an abscess cavity in the left lower lobe.

Fig. 8.—Left lower lobe removed from Dog Y10, experiment 21, thirteen days after embolism; a small abscess cavity is still present.

September 16, the dog appeared to be breathing more rapidly. A roentgenogram of the chest disclosed an increasing area of density with an area of rarefaction in the center.

September 18, a cavity formation about 1 cm. in diameter was present. The lead filing had dropped to the bottom of the cavity (fig. 7).

September 21, the dog did not appear very ill, and apparently was combating the infection successfully.

September 24, during the preparation for a lobectomy the dog died from the anesthesia.

Necropsy.—A firm mass about the size of a walnut could be felt in the left lower lobe. There were no pleural adhesions. On sectioning the lobe, a small abscess was encountered near the lower pole (fig. 8). Microscopically, a section taken through the abscess wall showed almost complete resolution of the inflammatory process. The lining wall of the abscess showed considerable fibrous tissue formation.

EXPERIMENT 22.—Dog Y11, weighing 6.7 Kg., Sept. 12, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was exposed and prepared for reception of the embolus in the routine manner. A culture of *Staphylococcus aureus* was implanted into a small metal tube, 4 mm. long and 2 mm. in diameter, and both ends of the tube were occluded with bits of muscle removed from the wound. The tube was introduced into the lumen of the vessel and forced onward with salt solution. The vein was ligated with silk and the skin approximated with a subcuticular stitch of silk. A roentgenogram of the chest localized the foreign body in the left lower lobe.

September 14, a roentgenogram of the chest revealed no evidence of a pathologic condition of the lung.

September 18, the roentgenogram disclosed for the first time definite signs of infiltration about the foreign body.

September 21, there was more evidence of infiltration in the area previously described.

September 28, the lung shadow present in the previous roentgenogram had largely subsided. The dog had remained well.

October 1, the lower left lobe was removed by operation. The specimen contained a small mass of fibrous tissue completely encasing the metal tube. No sections for microscopic examination were taken.

EXPERIMENT 23.—Dog Y12, weighing 10.5 Kg., Sept. 14, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was exposed and prepared for reception of the embolus. A cylinder of raw potato was cut with a glass tube and impregnated with a suspension of *Staphylococcus aureus*. The tube was inserted into the lumen of the vein and the embolus was forced onward with salt solution.

September 30, almost daily roentgenograms of the chest were taken but the lung remained clear.

EXPERIMENT 24.—Dog Y13, weighing 15.6 Kg., Sept. 14, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was exposed and prepared for reception of the embolus in the routine manner. A cylinder of raw potato was cut with a glass tube and impregnated with staphylococcus. The tube was inserted into the lumen of the vein and the embolus forced downward with salt solution. A small lead shot, which had been previously drilled into, was filled with a culture of *Staphylococcus aureus*, and the hole in the shot was plugged with a bit of muscle. This was likewise forced into the

circulation. The vein was closed with silk and the skin approximated with a subcuticular stitch of silk. A roentgenogram of the chest localized the lead shot in the left lower lobe.

September 16, a roentgenogram of the chest revealed suggestive infiltration about the lead shot.

September 18, the area of infiltration was quite extensive about the lead shot (fig. 9).

September 19, there appeared beginning cavitation in the center of the dense area.

September 23, the infiltration was subsiding but there was more evidence of cavity formation.

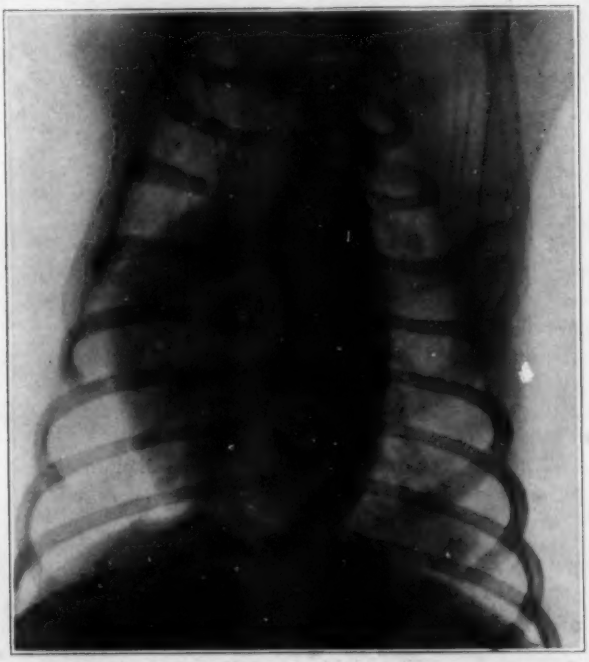

Fig. 9.—Dog Y13, experiment 24, four days after embolism; there is considerable infiltration about the lead shot with suggestive cavity formation.

September 26, the infiltration had largely subsided but the cavity was definitely present. The dog remained well.

September 28, the left lower lobe was removed by operation. On palpation a circumscribed area of increased density could be felt near the tip of the lobe. It seemed to be about the size of a walnut. On section the lead shot was found lying in a small abscess cavity. The small cylinder of potato was also found lying in a small abscess cavity a short distance away (fig. 10). Microscopically, a section taken through the abscess wall showed the lining to be composed of considerable fibrous tissue. Small cell infiltration extended for some distance into the adjoining lung tissue. A cellular exudate could be seen in the smaller bronchi.

EXPERIMENT 25.—Dog Y14, weighing 10 Kg., Sept. 15, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was

exposed and prepared for reception of the embolus in the routine manner. A piece of muscle taken from the wound was folded about a culture of *Staphylococcus aureus* and clamped with a small piece of paraffinized lead. The bit of muscle was then placed into a glass cannula, the cannula was inserted into the vein and the embolus was forced onward with salt solution. The vein was closed with silk and the skin approximated with a subcuticular stitch of silk. A roengenogram of the chest localized the foreign body in the left lower lobe.

September 18, a roentgenogram of the chest showed a beginning infiltration about the foreign body.

September 21, the shadow of density in the left lower lobe was quite marked (fig. 11).

Fig. 10.—Left lower lobe removed from Dog Y13, experiment 24, fourteen days after embolism; the cylinder of potato can be seen lying in a small abscess cavity: the adjoining lung tissues are infiltrated.

September 26, the area of infiltration was decreasing but a suggestive area of cavitation was present.

September 29, the lobe was removed by operation. The specimen showed a discolored wedge shaped area near the tip and gave evidence of a healing fibrotic infarct. Some induration could be felt beneath the scar in the lung tissue itself. Section through this area disclosed only fibrous tissue, which completely surrounded the piece of lead. Microscopically, a section taken through the area of infiltration showed the inflammatory process to be resolving. There was some fibrous tissue formation. The active inflammatory process extended for only a short distance into the adjoining lung tissue.

EXPERIMENT 26.—Dog Y15, weighing 6.4 Kg., Sept. 16, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was prepared in the usual manner. A piece of muscle removed from the wound was folded about a culture of *Staphylococcus aureus* and clipped together with a bit of paraffinized lead filing. The material was placed into a glass cannula, the cannula was inserted into the vein and the embolus forced onward with salt solution. The vein was ligated and the skin was closed in the usual manner. A roentgenogram of the chest localized the foreign body in the left lower lobe.

September 30, roentgenograms of the chest taken almost daily revealed the absence of any lesion in the lungs.

EXPERIMENT 27.—Dog Y16, weighing 10.2 Kg., Sept. 18, 1925, was given one-fourth grain of morphine. Under ether anesthesia the right jugular vein was

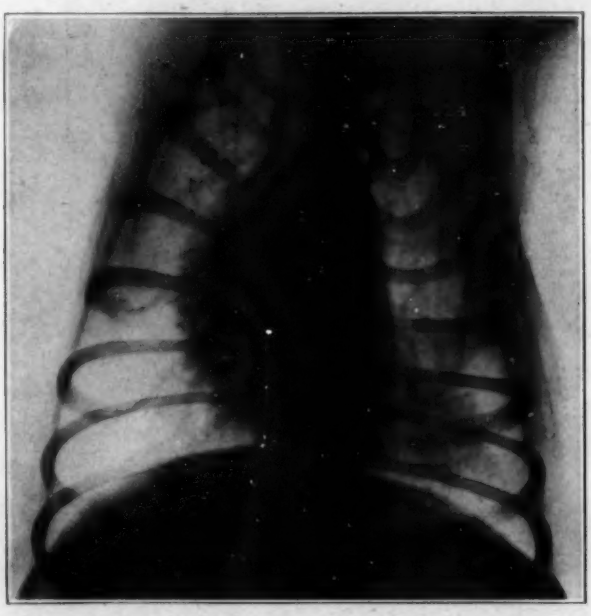

Fig. 11.—Dog Y14, experiment 25, six days after embolism; a large circumscribed area of density can be seen about the piece of lead in the left lower lobe.

prepared in the usual manner. A piece of human tonsil supplemented with cultures of *Staphylococcus aureus* and clipped with paraffinized lead filings was placed into the glass cannula, the cannula was inserted into the vein, and the embolus was forced onward with salt solution. The vein was sutured with silk and the skin was closed in the usual manner. A roentgenogram of the chest revealed the foreign body in the left lower lobe.

September 21, the dog appeared sick and had quite a tremor. When first seen in the morning there was blood in the cage, and examination revealed old blood in both nostrils. There was beginning infiltration in the left lower lobe (fig. 12).

September 23, the dog continued ill and would not eat. The entire left base cast a very dense shadow (fig. 13).

September 26, a roentgenogram of the chest showed a very dense shadow occupying the entire lower half of the left chest (fig. 14). The dog died at noon.

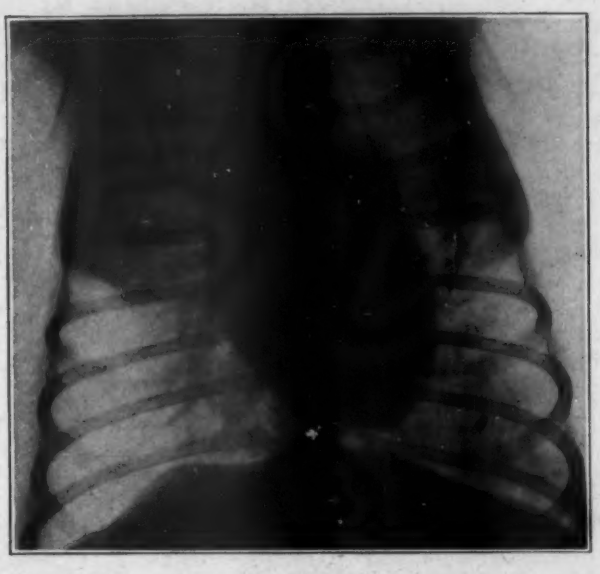

Fig. 12.—Dog Y16, experiment 27, three days after embolism; there is beginning infiltration about the piece of lead in the left lower lobe.

Fig. 13. Dog Y16, experiment 27, five days after embolism; there is marked consolidation of the left lower lobe.

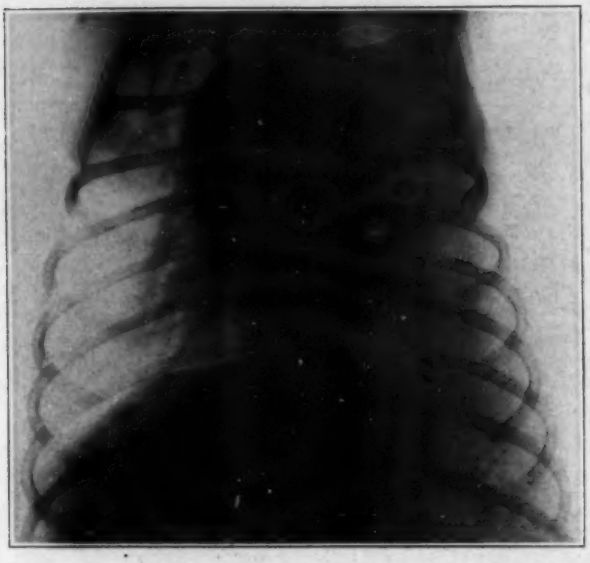

Fig. 14.—Dog Y16, experiment 27, eight days after embolism; the entire left lung casts a dense shadow.

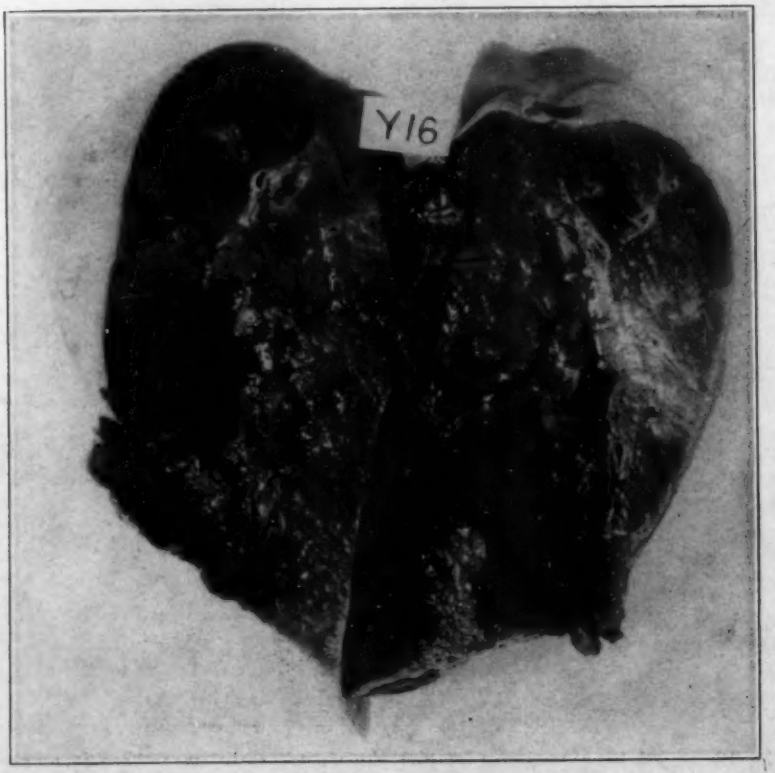

Fig. 15.—Left lower lobe removed at necropsy from Dog Y16, experiment 27, eight days after embolism; a large necrotic walled abscess is present.

Necropsy.—The pleural cavities contained a large amount of dark bloody fluid. There were no pleural adhesions. A large blood clot was present in the left pleural sinus. The entire left lower lobe was large and firm. In the lower part of the lobe an abscess cavity about the size of a walnut was found to have ruptured into the pleural cavity (fig. 15). The upper left lobe showed almost complete atelectasis while the lobes on the right were air containing and apparently normal. Microscopically, a section taken through the abscess wall showed an acute diffuse inflammation of the neighboring lung tissue. The infiltrating cells were mostly leukocytes. The inner wall of the abscess was composed of fibrin and necrotic tissue.

EXPERIMENT 28.—Dog Y12, weighing 10.5 Kg., Oct. 21, 1925, was given one-fourth grain of morphine. Under ether anesthesia the left jugular vein was prepared in the usual manner. The right femoral vein was then isolated and a segment removed. A portion of this segment of vein was prepared according to the technic previously described and inoculated with a pure culture of *Staphylococus aureus.* The embolus was then placed into a glass cannula and injected into the jugular vein. The skin incisions were closed with a subcuticular stitch of silk. A roentgenogram of the chest localized the foreign body in the right lower lobe.

October 23, there was no roentgenographic evidence of infiltration about the foreign body.

October 26, no definite evidence of a pulmonary lesion could be determined.

November 10, the dog remained well and at no time did a pulmonary lesion develop.

EXPERIMENT 29.—Dog Y18, weighing 16.3 Kg., Oct. 2, 1925, was given one-fourth grain of morphine. Under ether anesthesia the left jugular vein was prepared in the usual manner. A segment of femoral vein removed from Dog Y15 was inoculated with cultures of *Bacillus coli* and *Staphylococcus aureus* and injected into the jugular vein. The vessel was closed with silk and the skin was approximated with a subcuticular stitch of silk. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 5, a roentgenogram of the chest revealed considerable infiltration about the foreign body in the left lower lobe (fig. 16).

October 7, the infiltration was quite marked and extensive (fig. 17).

October 12, a small area of rarefaction appeared in the central portion of the dense area. It was decided to perform a lobectomy. The dog appeared quite well. Examination of the left lower lobe revealed a fibrotic healed infarct. A small area of induration could be felt within the central portion of the lobe. When the lobe was sectioned a small abscess cavity was encountered (fig. 18). Microscopically, a section taken through the abscess wall showed the inner wall to be made up of fibrous tissue. Histologically, the picture was one of resolving inflammation.

EXPERIMENT 30.—Dog Y19, weighing 16.4 Kg., Oct. 3, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment from the right femoral vein was removed, inoculated with cultures of *Bacillus coli* and *Staphylococcus aureus* and introduced into the left jugular vein. The vessel was sutured with silk and both skin incisions were closed with a subcuticular stitch of silk. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 5, there was beginning infiltration about the foreign body.

October 7, infiltration was marked and beginning cavitation was apparent.

October 9, a well defined cavity was present in the roentgenogram.

Fig. 16.—Dog Y18, experiment 29, three days after embolism; there is beginning infiltration about the embolus in the left lower lobe.

Fig. 17.—Dog Y18, experiment 29, five days after embolism; a dense circumscribed area is present about the embolus.

October 12, an abscess cavity about 2 cm. in diameter could be seen (fig. 19.)

November 1, roentgenograms of the chest had shown a gradual subsidence of the infiltration. The cavity likewise had decreased in size.

November 6, the left lower lobe was removed by operation. There were numerous pleural adhesions attaching the lobe to the diaphragm. A small area of induration could be felt within the lobe. When it was sectioned a small fibrous walled cavity containing the bits of lead and silk was encountered (fig. 20). Microscopically, a section taken through the abscess wall showed the inner wall of the cavity to be made up almost entirely of fibrous tissue. There was still considerable round cell infiltration.

Fig. 18.—Left lower lobe removed from Dog Y18, experiment 29, ten days after embolism; a small abscess cavity containing the piece of lead is still present.

EXPERIMENT 31.—Dog Y20, weighing 10.6 Kg., Oct. 3, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment from the right femoral vein was removed, inoculated with cultures of *Bacillus coli* and *Staphylococcus aureus* and introduced into the left jugular vein. The vessel was sutured with silk and both skin incisions approximated with a subcuticular stitch of silk. A roentgenogram of the chest localized the foreign body in the left lower lobe (fig. 21).

October 5, a roentgenogram of the chest showed considerable infiltration about the foreign body in the left lower lobe (fig. 22).

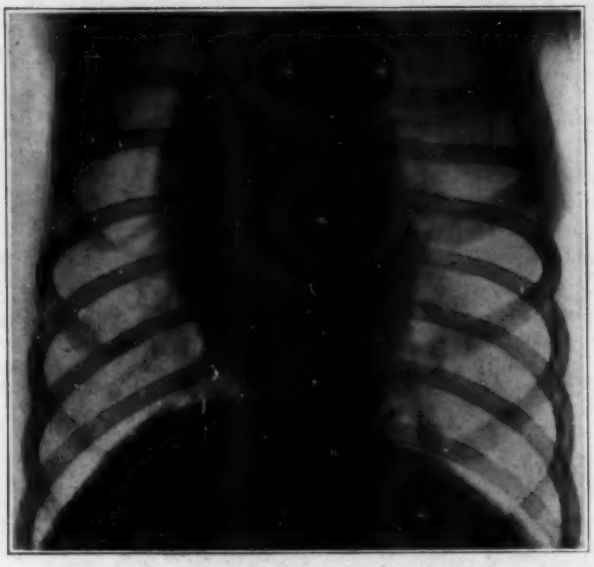

Fig. 19.—Dog Y19, experiment 30, nine days after embolism; a well defined abscess cavity is present in the left lower lobe.

Fig. 20.—Left lower lobe removed from Dog Y19, experiment 30, thirty-three days after embolism; a small fibrous walled cavity is still present.

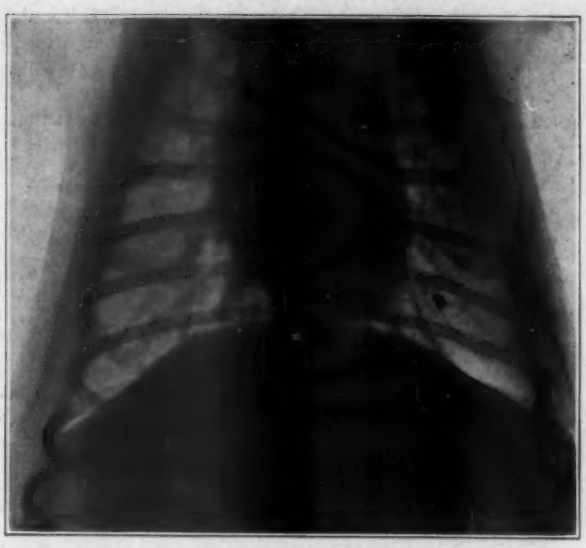

Fig. 21.—Dog Y20, experiment 31, immediately after embolism; the piece of lead indicates the position of the embolus in the left lower lobe.

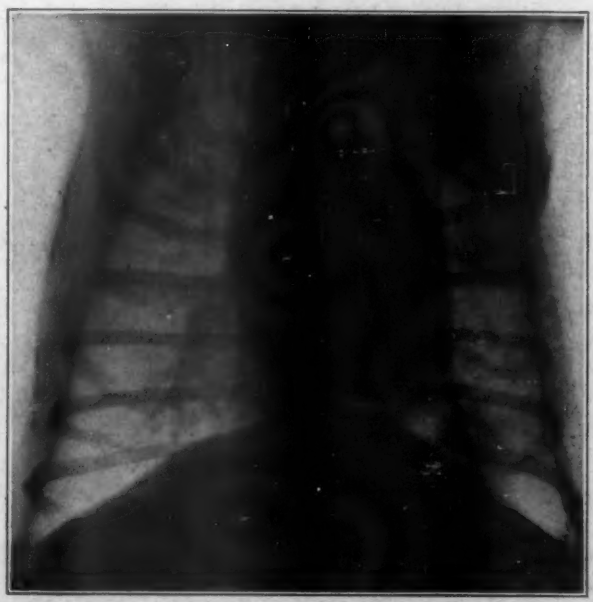

Fig. 22.—Dog Y20, experiment 31, two days after embolism; there is beginning infiltration about the embolus.

October 7, a large circumscribed area of density was present in the left lower lobe.

October 9, a roentgenogram showed a large cavity in the center of the area of density. The lead filing had dropped to the bottom of the cavity (fig. 23). The lobe was removed by operation. The left lower lobe revealed an indurated mass in the center of the lobe. Fluctuation within the mass could be palpated. When it

Fig. 23.—Dog Y20, experiment 31, six days after embolism; a large abscess cavity is present; the lead filings have dropped to the bottom of the cavity.

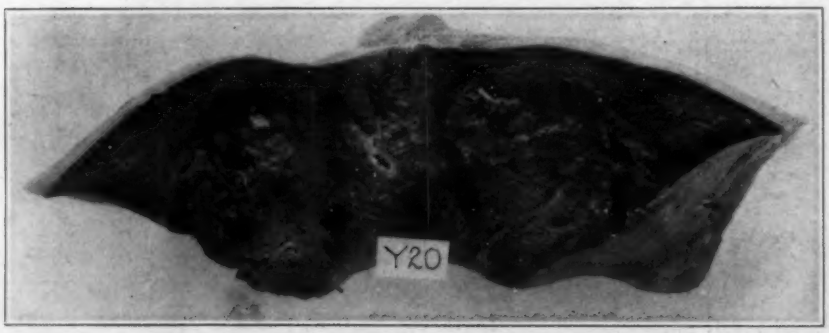

Fig. 24.—Left lower lobe removed from Dog Y20, experiment 31, six days after embolism; a large necrotic walled abscess is present.

was sectioned an abscess cavity about the size of a walnut was encountered containing necrotic tissue and pus (fig. 24). Cultures taken from the pus revealed staphyloccoccus and *Bacillus coli*. Microscopically, a section taken through the abscess wall showed the inner wall of the cavity to be made up of fibrin and necrotic tissue. The adjoining lung tissue was densely infiltrated with leuko-

cytes. There was considerable recent hemorrhage. Histologically, the picture was one of acute diffuse inflammation.

EXPERIMENT 32.—Dog Y21, weighing 15 Kg., Oct. 7, 1925, was given one-fourth grain of morphine. Under ether anesthesia a section from the right femoral vein was removed, inoculated with cultures of *Bacillus coli* and introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 8, an area of cloudiness could be seen about the foreign body.

October 12, the area of density was larger in size and a distinct cavity was visible (fig. 25).

November 3, subsequent roentgenograms of the chest revealed a persistence of the cavity.

November 9, the left lower lobe was removed by operation. The specimen showed a healed fibrotic lesion (fig. 26). Microscopic sections were not taken.

EXPERIMENT 33.—Dog Y22, weighing 20 Kg., Oct. 7. 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of right femoral vein removed from Y21 and inoculated with cultures of *Bacillus coli* was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 8, there was an area of beginning density about the foreign body.

October 12, considerable infiltration could be seen in the left lower lobe.

October 16, the infiltration had almost subsided.

October 19, a roentgenogram of the chest showed the lungs to be clear.

October 22, the dog was killed in a fight.

Necropsy.—The pleural cavities were free of fluid and adhesions. The lungs were normal except for an area of fibrosis completely encasing the bit of lead. Microscopically the section showed almost complete fibrotic organization. A few round cells were still present.

EXPERIMENT 34.—Dog Y23, weighing 11.3 Kg., Oct. 14, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of the right femoral vein was removed, inoculated with cultures of *Bacillus coli, Staphylococcus aureus* and streptococcus, and introduced into the right jugular vein. A roentgenogram of the chest localized the foreign body in the right upper lobe.

October 17, a roentgenogram of the chest showed considerable infiltration about the foreign body.

October 19, there was a consolidation of the entire upper right lobe. The dog appeared ill.

October 23, the dog continued ill and would not eat. The right middle lobe appeared consolidated. While the animal was being placed on the roentgen-ray table, considerable bloody and purulent sputum appeared in the nostrils on coughing and sneezing. The infiltration appeared less dense.

October 26, there remained about the same degree of density in the right lung.

October 28, bronchoscopic examination revealed mucopurulent material in the trachea apparently arising from the bronchi on the right side.

October 31, a roentgenogram of the chest showed less density of the right upper lobe. The middle was fairly clear. The dog began to eat better.

November 9, there was almost complete resolution of the pneumonic process.

December 11, the animal was sacrificed.

Necropsy.—There were no pleural adhesions. The lungs appeared normal except for some fibrosis of the right upper lobe. The piece of lead was recovered in an area of fibrosis.

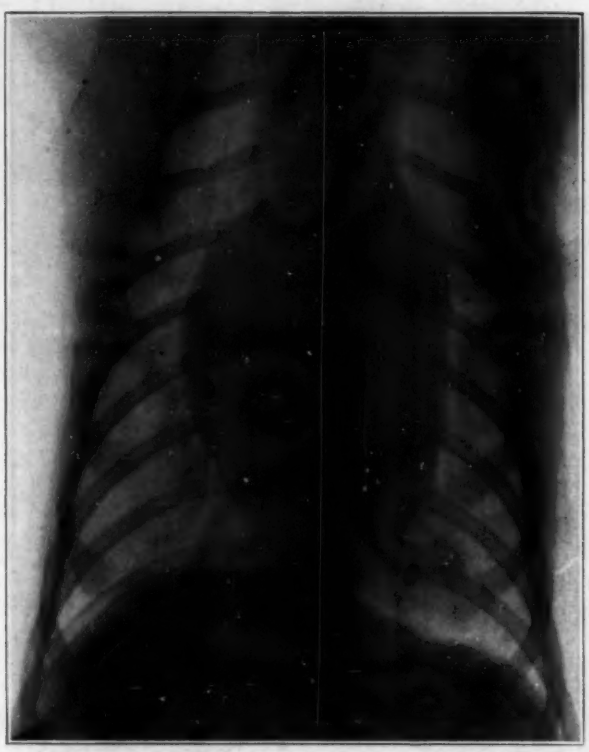

Fig. 25.—Dog Y21, experiment 32, five days after embolism; cavity formation about the embolus is quite distinct.

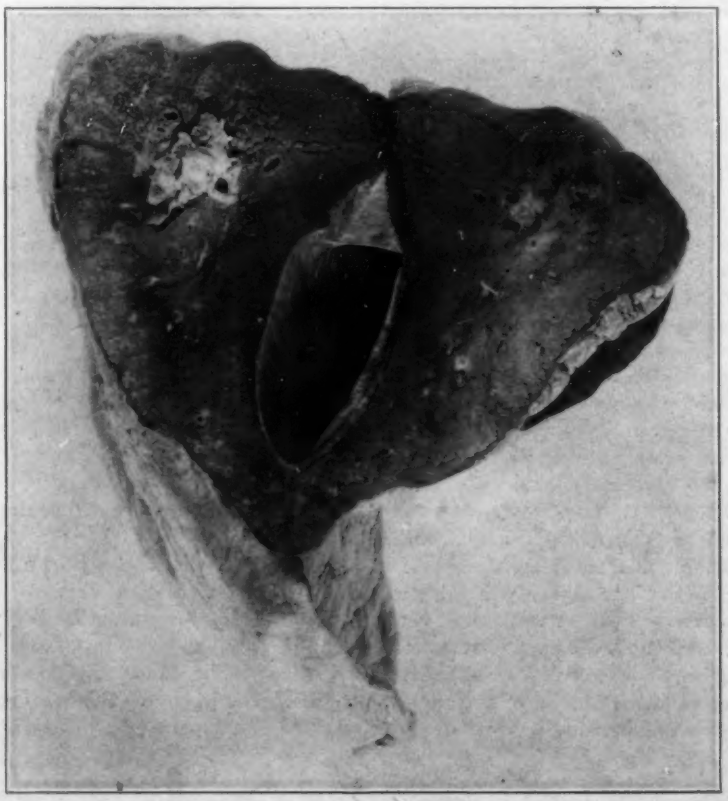

Fig. 26.—Left lower lobe removed from Dog Y21, experiment 32, thirty-three days after embolism; a healed area of fibrosis is present.

EXPERIMENT 35.—Dog Y24, weighing 13.7 Kg., Oct. 14, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of femoral vein, removed from Y23 and inoculated with cultures of *Bacillus coli, Staphylococcus aureus* and streptococcus, was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

October 15, a roentgenogram of the chest showed beginning infiltration about the foreign body.

October 17, there was beginning cavitation in the area of density.

November 16, frequent roentgenograms of the chest had shown the persistence of a small cavity in a zone of infiltration in the right lower lobe. It was decided to remove the lobe by operation. The specimen removed showed considerable fibrosis in an area surrounding the lead filing with almost complete obliteration of a small cavity. Microscopically, the section showed complete fibrotic healing.

EXPERIMENT 36.—Dog Y25, weighing 14.5 Kg., Oct. 16, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment was removed from the right femoral vein, inoculated with cultures of pneumococcus and streptococcus, and introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 19, roentgenograms of the chest had not shown any infiltration.

October 22, there was a moderate degree of infiltration about the foreign body.

November 3, the subsequent roentgenograms had shown nothing further and the infiltration had subsided. The dog's general condition remained good.

December 1, the dog was killed in a fight.

Necropsy.—Both pleural cavities and lungs were normal. The lead filing was recovered in the left lower lobe encased in a small amount of fibrous tissue.

EXPERIMENT 37.—Dog Y26, weighing 9.5 Kg., Oct. 16, 1925, was given one-sixth grain of morphine. Under ether anesthesia a segment of femoral vein, removed from Y25 and inoculated with pneumococcus and streptococcus, was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 17, a roentgenogram of the chest showed a diffuse cloudiness of the left lower lobe.

October 19, density of a moderate degree was confined to the left lower lobe. The dog appeared ill.

October 22, the consolidation was subsiding.

October 27, the lungs were clear. The animal was allowed to live.

EXPERIMENT 38.—Dog Y27, weighing 13.6 Kg., Oct. 21, 1925, was given one-fourth grain of morphine. A segment of femoral vein, removed from Y12 and inoculated with cultures of *Staphylococcus aureus,* was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

October 26, a roentgenogram of the chest showed a large area of infiltration about the foreign body with a definite beginning cavitation in the central portion.

October 31, a small abscess cavity in the right lower lobe on a level with the dome of the diaphragm was visible (fig. 27).

November 9, the process had subsided but a small cavity apparently remained with only a slight degree of infiltration surrounding.

November 17, the right lower lobe was removed by operation. A few pleural adhesions existed between the lobe and the diaphragm. Sectioning the lung revealed a large abscess cavity (fig. 28). Microscopically, a section taken through

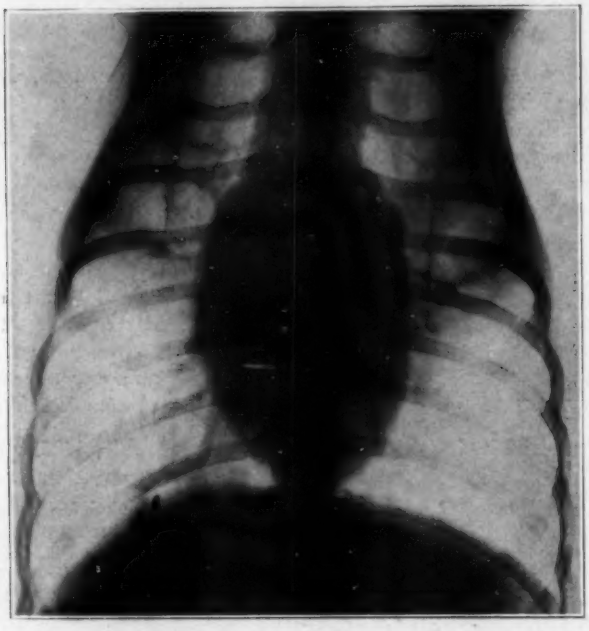

Fig. 27.—Dog Y27, experiment 38, ten days after embolism; a small abscess cavity is visible in the right lower lobe.

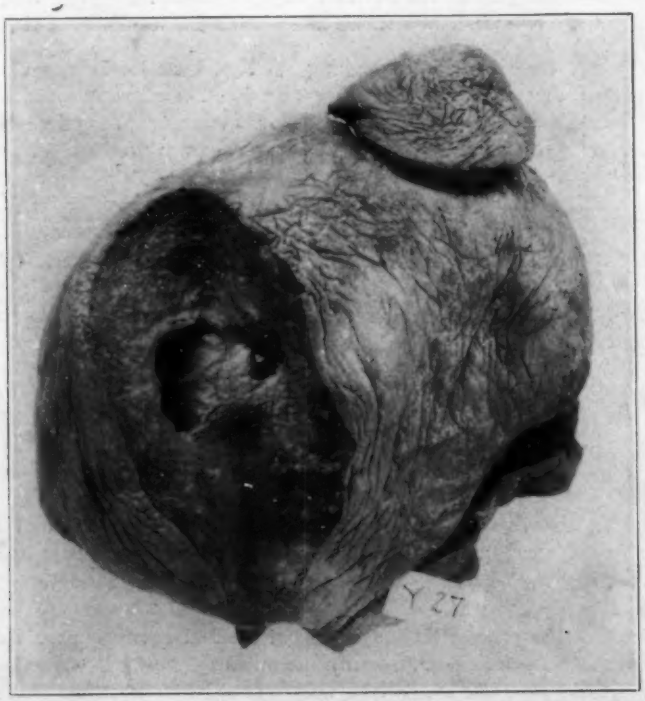

Fig. 28.—Right lower lobe removed from Dog Y27, experiment 38, twenty-seven days after embolism; a fibrous lined cavity is still present.

the abscess wall showed the cavity lined with a thin zone of fibrous tissue. There was very little cellular exudate.

EXPERIMENT 39.—Dog Y28, weighing 8.2 Kg., Oct. 21, 1925, was given one-sixth grain of morpine. Under ether anesthesia a segment of vein, removed from Y12 and inoculated with *Staphylococcus aureus,* was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 23, a roentgenogram of the chest did not reveal any infiltration about the foreign body in the left lower lobe.

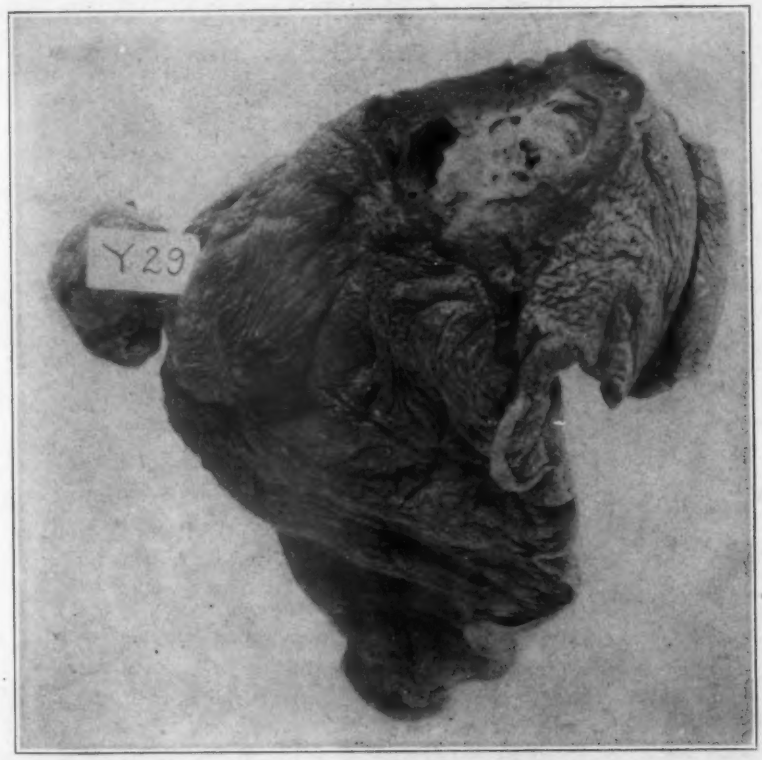

Fig. 29.—Left lower lobe removed from Dog Y29, experiment 40, twenty-three days after embolism; a small fibrous lined cavity can be seen.

November 3, repeated roentgenograms continued to show normal lung fields.

November 9, the dog died from distemper.

Necropsy.—The lungs showed patches of grayish and reddish areas on cut section. Pus exuded from the smaller bronchi. The piece of lead was recovered surrounded by a small area of fibrosis.

EXPERIMENT 40.—Dog Y29, weighing 11.5 Kg., Oct. 26, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment was removed from the right femoral vein, inoculated with cultures of a long chain nonhemolytic streptococcus and introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 28, there was beginning infiltration about the foreign body.

October 31, a definite abscess cavity could be seen in the left lower lobe.

November 9, the abscess cavity was quite large. The lead filing could be seen in the bottom of the cavity.

November 18, the left lower lobe was removed by operation. The lobe was not bound down by pleural adhesions. A moderately large indurated mass could be felt. When sectioned a fairly large abscess cavity was encountered (fig. 29). Microscopically, a section taken through the abscess wall showed a lining membrane of fibrous tissue. Histologically, the picture was one of an almost completely healed process.

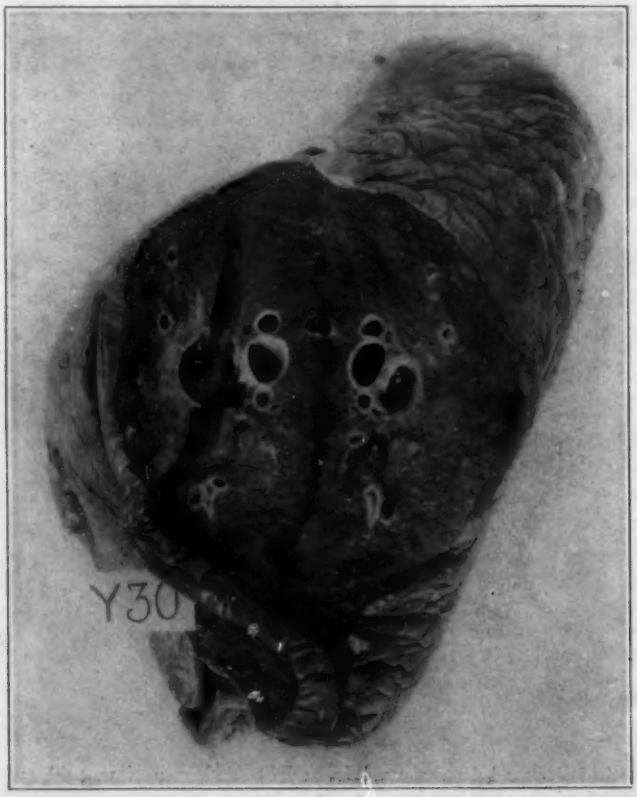

Fig. 30.—Right lower lobe removed from Dog Y30, experiment 41, sixteen days after embolism; a small abscess cavity can be seen.

EXPERIMENT 41.—Dog Y30, weighing 9.5 Kg., Oct. 26, 1925, was given one-sixth grain of morphine. Under ether anesthesia a segment of femoral vein, removed from Y29 and inoculated with cultures of a long chain streptococcus, was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

November 11, the dog failed to show any definite symptoms indicative of an infection. Inability to obtain good roentgengrams on repeated occasions compelled us to remove the right lower lobe by operation in order to ascertain the nature of the lesion. On its external surface the lobe appeared to be normal. Near the tip of the lobe a small abscess cavity was found after sectioning (fig. 30).

Microscopically, a section taken through the abscess wall showed a fibrous tissue lining with very little cellular infiltration.

EXPERIMENT 42.—Dog Y31, weighing 8 Kg., Oct. 26, 1925, was given one-sixth grain of morphine. Under ether anesthesia a segment of vein, removed from Y29 and inoculated with cultures of a long chain streptococcus, was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

October 28, beginning infiltration could be seen about the foreign body.

October 31, the infiltration was more marked.

November 4, a definite multiloculated abscess cavity with a peripheral zone of infiltration was visible.

November 9, the cavity was visible through the apex of the heart shadow. The piece of lead had assumed the most dependent portion of the cavity.

November 18, the abscess persisted in size and the lobe was removed by operation. A definite thin walled cavity could be seen through the visceral pleura (fig. 31). Section through the lesion revealed a multiloculated thin walled cavity. Microscopically, a section through the wall showed a smooth fibrous tissue inner lining (fig. 32). A few round cells were present in the narrow abscess wall. The process was almost healed.

EXPERIMENT 43.—Dog Y32, weighing 13.1 Kg., Oct. 31, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of the femoral vein removed from Y33 and inoculated with a culture of pneumococcus type II was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

November 9, the dog remained well. Daily roentgenograms of the chest revealed no developmental lesion.

November 15, the dog contracted distemper from which he died. Signs of illness preceded death for only a few days.

Necropsy.—The lungs and bronchi showed the characteristic distemper findings. There were scattered patches of consolidation throughout both lungs. Pus exuded from the bronchi. The lead filing was encased in a small amount of fibrous tissue.

EXPERIMENT 44.—Dog Y33, weighing 10.1 Kg., Oct. 31, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of the right femoral vein inoculated with a culture of pneumococcus type II was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

November 19, repeated roentgenograms failed to show any developmental lesion, although the dome of the diaphragm always obscured the area occupied by the foreign body. The lobe was removed by operation. The removed lobe was normal. The piece of lead was encased in a slight amount of fibrous tissue. Microscopically the section showed dense fibrous tissue with a few infiltrating round cells.

EXPERIMENT 45.—Dog Y34, weighing 6.2 Kg., Oct. 31, 1925, was given one-sixth grain of morphine. Under ether anesthesia a segment of the femoral vein, removed from Y33 and inoculated with a culture of pneumococcus type II, was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

November 12, repeated roentgenograms disclosed no developmental lesion in the lung.

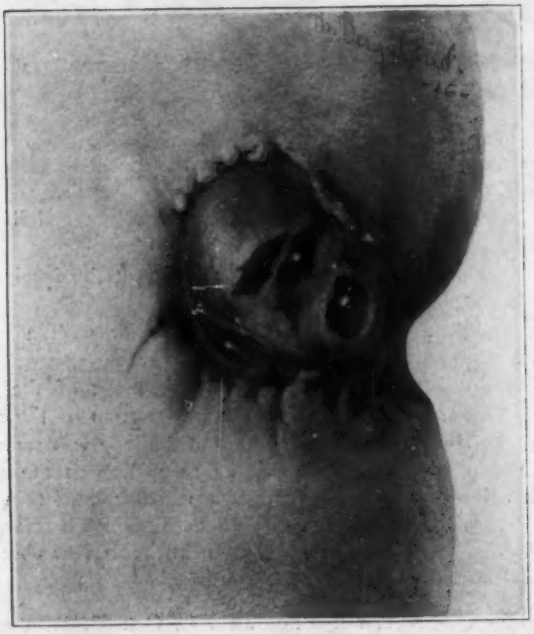

Fig. 31.—Exterior appearance of the abscess cavity in the left lower lobe removed from Dog Y31, experiment 42, twentw-three days after embolism.

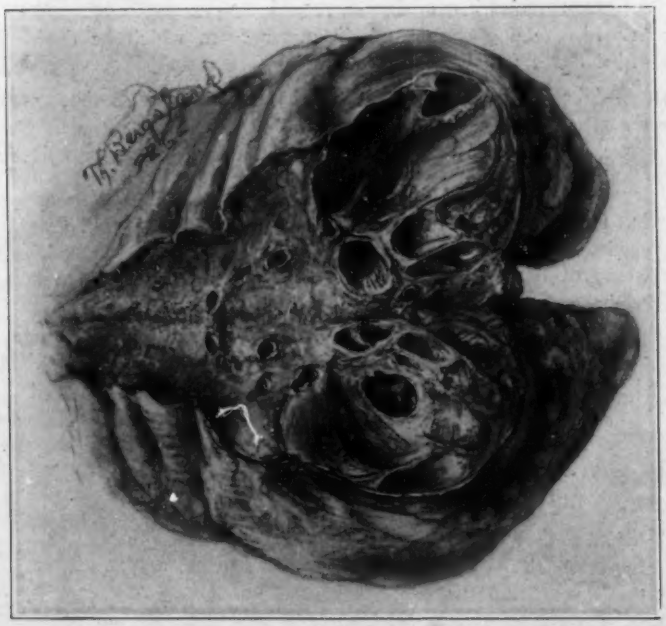

Fig. 32.—Appearance after sectioning the removed lobe of Dog Y31, experiment 42; the multiloculated character of the abscess cavity should be noted.

November 14, the dog was killed because of a developing distemper.

Necropsy.—The typical distemper appearance of the lungs and bronchi was found. The lead filing was encased in fibrous tissue.

EXPERIMENT 46.—Dog Y35, weighnig 22.6 Kg., Nov. 2, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of the femoral vein, removed from Y37 and inoculated with cultures of pneumococcus type II and *Staphylococcus aureus,* was introduced into the left jugular vein. A roentgenogram of the chest revealed the foreign body in the right lower lobe.

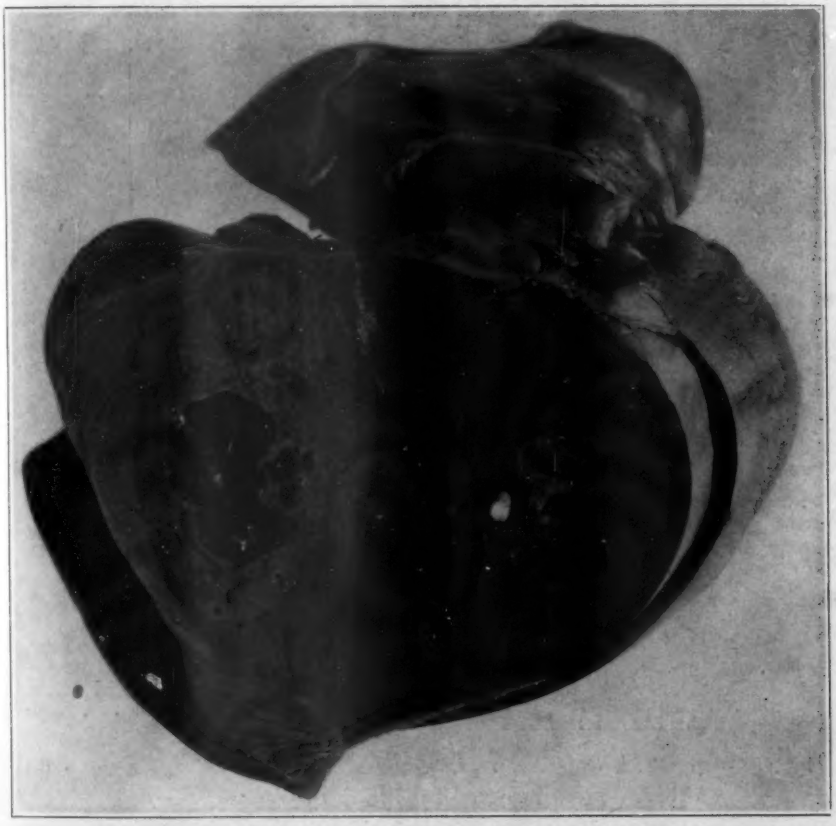

Fig. 33.—Right lower lobe removed from Dog Y35, experiment 46, twenty-two days after embolism; a large abscess cavity is present.

November 5, there was beginning infiltration about the foreign body.

November 12, a small cavity was seen in the zone of infiltration.

November 17, a cavity measuring approximately 2 cm. in diameter could be seen.

November 24, the dog was killed in a fight.

Necropsy.—A fairly large indurated mass could be felt in the right lower lobe. When sectioned, an abscess cavity with numerous secondary cavities was encountered (fig. 33). Microscopically, a section taken through the abscess wall showed

a fibrotic smooth walled cavity. There was a considerable zone of round cell infiltration.

EXPERIMENT 47.—Dog Y36, weighing 13.2 Kg., Nov. 2, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of the right femoral vein was removed, inoculated with cultures of pneumococcus type II and *Staphylococcus aureus,* and introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

November 5, there was beginning infiltration about the foreign body.

November 9, a large cavity could be seen about the foreign body (fig. 34).

Fig. 34.—Dog Y36, experiment 47, seven days after embolism; a large thin walled cavity without a surrounding lung infiltration can be seen.

November 17, the cavity had increased in size. The dog appeared ill.

November 21, the dog continued ill and showed symptoms of distemper. A roentgenogram revealed a large cavity in the right lower lobe and a diffuse cloudiness in the region of the right middle lobe, evidently the picture of a distemper lung (fig. 35).

November 23, the right lower lobe was removed by operation. A consolidated right middle lobe was also removed. The right lower lobe was definitely increased in size. Induration could be felt within. When sectioned a large abscess cavity was found (fig. 36). Microscopically, a section taken through the abscess wall showed a fairly smooth fibrotic lined cavity. There was a moderate degree of cellular infiltration.

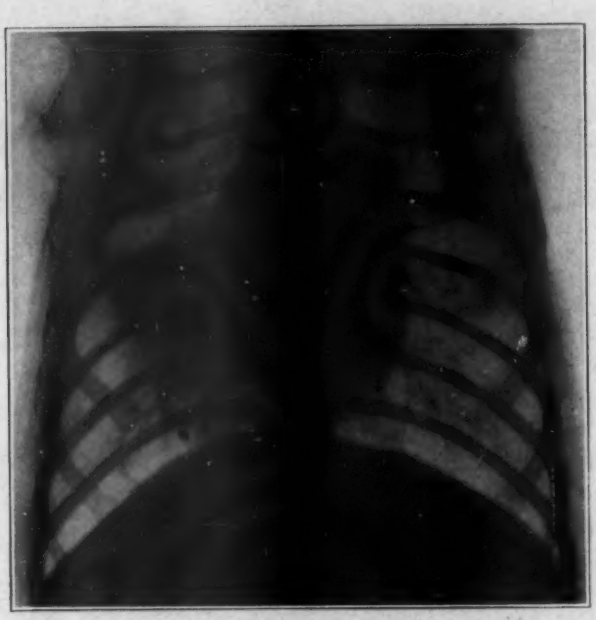

Fig. 35.—Dog Y 36, experiment 47, nineteen days after embolism; the abscess cavity in the right lower lobe is larger; the right middle lobe is densely consolidated.

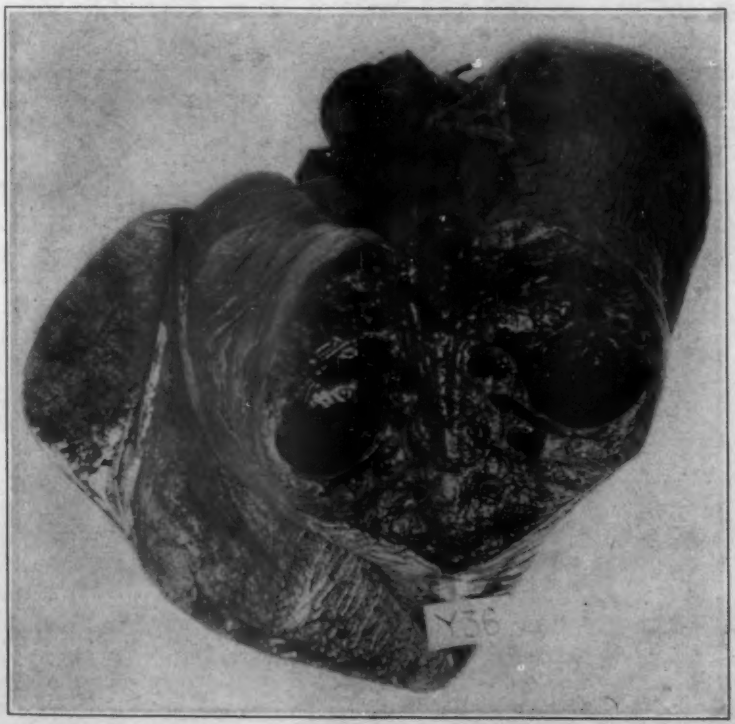

Fig. 36.—Right lower lobe removed from Dog Y36, experiment 47, twenty-one days after embolism; a large abscess cavity is present.

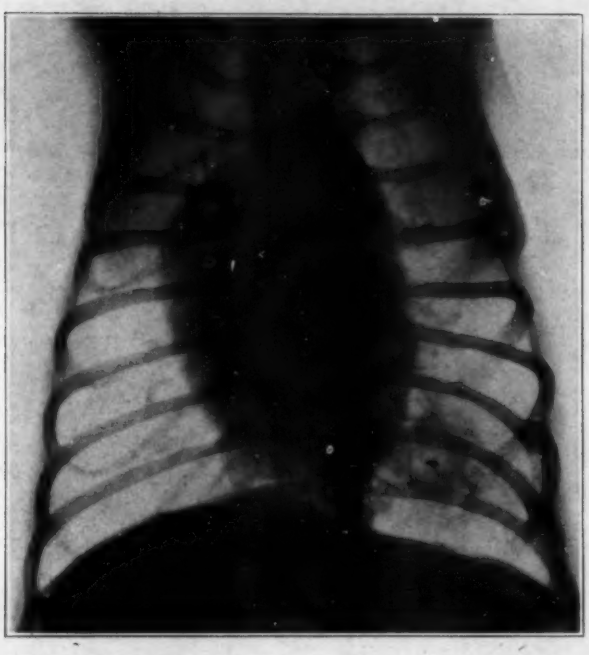

Fig. 37.—Dog Y38, experiment 49, eight days after embolism; an abscess cavity can be seen in the left lower lobe.

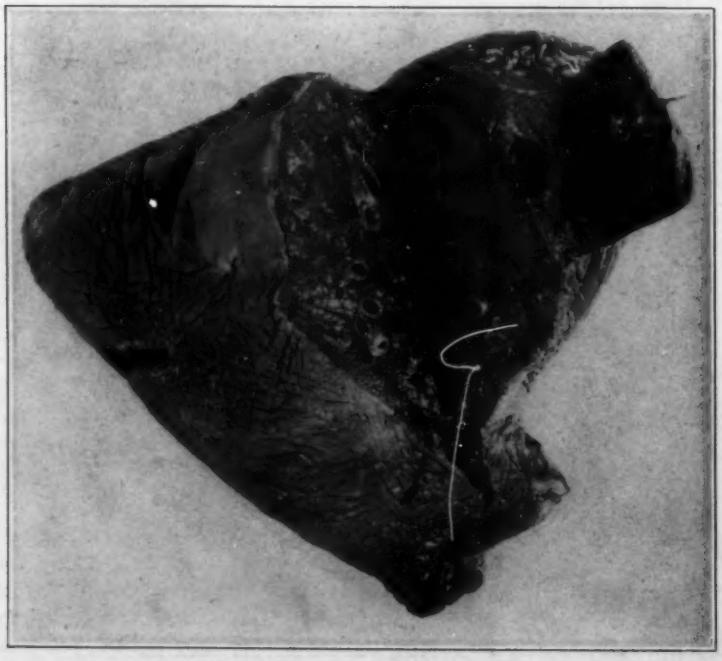

Fig. 38.—Left lower lobe removed from Dog Y38, experiment 49, sixteen days after embolism; a small abscess cavity is still present.

EXPERIMENT 48.—Dog Y37, weighing 18.9 Kg., Nov. 2, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of the femoral vein removed from Y35 and inoculated with cultures of pneumococcus type II and *Staphylococcus aureus*, was introduced into the left jugular vein. A roentgenorgam of the chest localized the foreign body in the right lower lobe.

November 5, the lungs appeared normal.

November 9, some infiltration could be seen about the foreign body.

November 13, the dog appeared well. The infiltration remained about the same.

November 19, the infiltration had largely subsided. No cavity formation had appeared. It was decided to remove the lobe by operation. The removed lobe appeared normal. The piece of lead was recovered encased in fibrous tissue.

EXPERIMENT 49.—Dog Y38, weighing 12.1 Kg., Nov. 11, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment was removed from the right femoral vein, inoculated with cultures of *Staphylococcus aureus, Bacillus coli* and pneumococcus type II, and introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

November 12, beginning infiltration was apparent about the foreign body.

November 14, infiltration of the left lower lobe was quite marked. Cavitation had appeared in the central portion. The dog appeared slightly ill.

November 19, quite a marked area of cavitation was present (fig. 37).

November 24, cavitation was well marked. The foreign body had dropped to the bottom of the cavity.

November 27, the infiltration had subsided and the cavity had decreased considerably in size. The left lower lobe was removed by operation. In the removed lobe an area of induration could be felt near the tip of the lobe. The visceral pleura was considerably thickened. On section thick indurated lung tissue was encountered. A small abscess cavity was still present (fig. 38). Microscopically, a section taken through the abscess wall showed the lining of the cavity to be composed chiefly of fibrous tissue. There was considerable cell exudation still present.

EXPERIMENT 50.—Dog Y39, weighing 14.8 Kg., Nov. 11, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of the femoral vein, removed from Y38 and inoculated with cultures of *Staphylococcus aureus, Bacillus coli* and pneumococcus type II, was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

November 13, there was beginning infiltration about the foreign body in the right lower lobe.

November 16, considerable infiltration was present in the right lower lobe.

November 21, a well marked cavity could be seen within the area of density.

November 23, the right lower lobe was removed by operation. The removed lobe was considerably increased in size and an indurated mass could be felt within. Section through this mass revealed a large, necrotic walled abscess (fig. 39). Microscopically, a section taken through the abscess wall showed a fairly smooth fibrous lined cavity containing necrotic and fibrous tissue. There was a moderate degree of diffuse inflammation.

EXPERIMENT 51.—Dog Y40, weighing 17 Kg., Nov. 11, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of femoral vein removed from Y38 and inoculated with cultures of *Staphylococcus aureus, Bacillus coli* and pneumococcus type II, was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

November 12, there was beginning infiltration about the foreign body

November 14, the infiltration was more marked in extent.

November 16, beginning cavitation was seen within the area of density.

November 19, the infiltration was subsiding. Signs of cavitation had disappeared.

November 24, the infiltration was very slight. The lesion had apparently resolved. The lobe was removed by operation. The removed lobe presented a normal appearance. There was a small area of fibrosis about the lead. Microscopically, the section showed dense fibrous tissue formation. There was still some cellular exudate present.

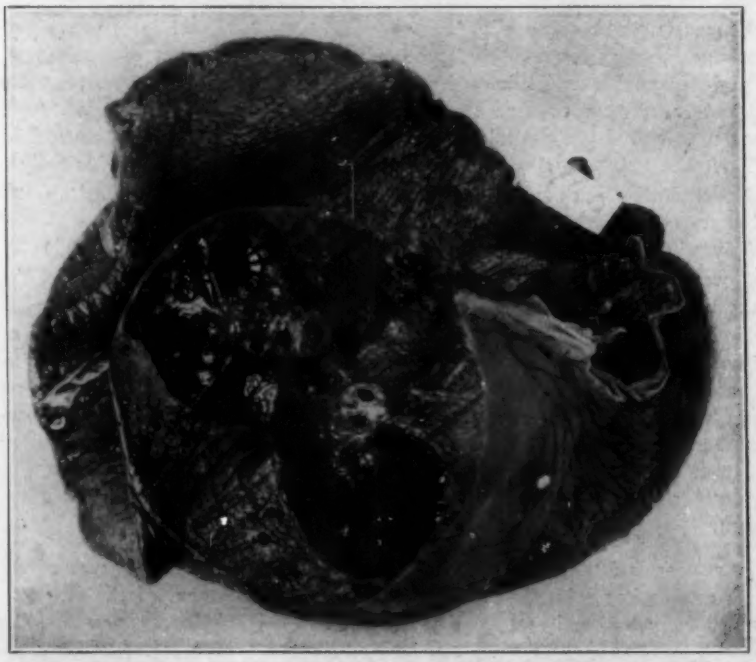

Fig. 39.—Right lower lobe removed from Dog Y39, experiment 50, twelve days after embolism; a large abscess cavity is present.

EXPERIMENT 52.—Dog Y48, weighing 13 Kg., Dec. 14, 1925, was given one-fourth grain of morphine. Under ether anesthesia a segment of the right femoral vein was removed, inoculated with cultures of *Staphylococcus aureus, Bacillus coli,* streptococcus and pneumococcus, and introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the right lower lobe.

December 15, there was beginning infiltration about the foreign body.

December 16, the infiltration was more marked.

December 17, within an increasing area of density beginning rarefaction could be seen.

December 19, the infiltration was subsiding. The signs of cavitation were distinctly visible.

December 21, the lesion was resolving.

Jan. 14, 1926, the lesion had resolved.

EXPERIMENT 53.—Dog Y51, weighing 8.5 Kg., Jan. 13, 1926, was given one-fourth grain of morphine. Under ether anesthesia a segment of right femoral vein was removed, inoculated with pneumococcus type I, *Staphylococcus aureus, Bacillus coli* and streptococcus, and introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

January 14 there was beginning infiltration about the foreign body.

January 16, the infiltration was more marked. Beginning cavitation was apparent.

January 21, the dog showed pronounced dyspnea. He appeared to be quite ill. When the chest wall was percussed a hyperresonant note was elicited throughout. A roentgenogram of the chest revealed a collapse of the entire left lung with no evidence of fluid. The abscess had apparently ruptured into the pleural cavity giving rise to a pneumothorax.

January 23, the dog appeared quite well. Signs of pneumothorax were still present. Difficulty was experienced in obtaining good roentgenograms on account of the rapid breathing. There was no evidence of fluid.

January 25, the dog was found dead in his cage.

Necropsy.—There was a small amount of pleural fluid. The left lower lobe was bound firmly to the mediastinum near the hilum of the lung. When air was forced into the bronchial tree through the trachea, air could be heard escaping in the region of the adhesions in the left lower lobe. The lungs could not be inflated with air. The lungs were normal, except for an indurated mass in the left lower lobe lying next to the pleural adhesions. Suppurating mediastinal and bronchial glands were found. On section the mass was seen to consist of inflammatory tissue. The abscess cavity had apparently been obliterated from partial rupture into the pleural cavity and mediastinum and the resultant collapse of the lung.

EXPERIMENT 54.—Dog Y52, weighing 6.6 Kg., Jan. 13, 1926, was given one-sixth grain of morphine. Under ether anesthesia a segment of femoral vein, removed from Y51 and inoculated with cultures of *Staphylococcus aureus, pneumoccocus* type I, *Bacillus coli* and streptococcus, was introduced into the left jugular vein. A roentgenogram of the chest localized the foreign body in the left lower lobe.

January 16, there was considerable infiltration in the area about the foreign body.

January 19, the dog appeared quite ill.

January 21, the dog was found dead in his cage.

Necropsy.—Both pleural cavities contained a cloudy hemorrhagic fluid. Numerous pleural adhesions plastered the left lower lobe to the lateral chest wall. An abscess, perforating into the pleural cavity, was found in this lobe. Cultures from the pus recovered the organisms placed into the embolus.

EXPERIMENT 55.—Dog Y59, weighing 13.6 Kg., March 18, 1926, was given one-fourth grain of morphine. Under procain anesthesia the femoral vein was exposed and a portion central to the point of division in the lower leg was removed. The segment of vein was inoculated with cultures of *Bacillus coli* and streptococcus and the prepared embolus was injected into the upper portion of the femoral vein. The vein was ligated and the wound closed with silk sutures. A roentgenogram of the chest localized the foreign body in the left lower lobe.

March 19, the animal appeared ill and would not eat. The rectal temperature was 40.2 C. A roentgenogram of the chest showed a large area of circumscribed infiltration about the piece of lead in the left lower lobe with a beginning central area of rarefaction (fig. 40).

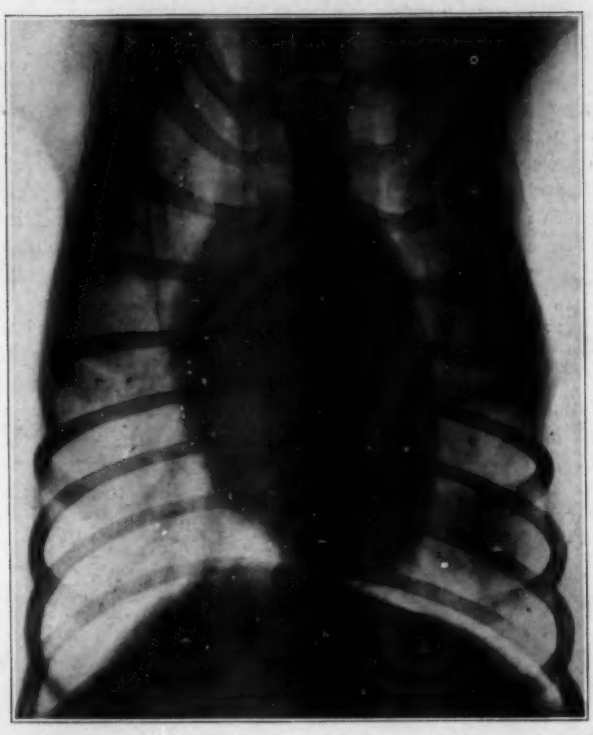

Fig. 40.—Dog Y59, experiment 55, one day after embolism; there is beginning infiltration about the embolus.

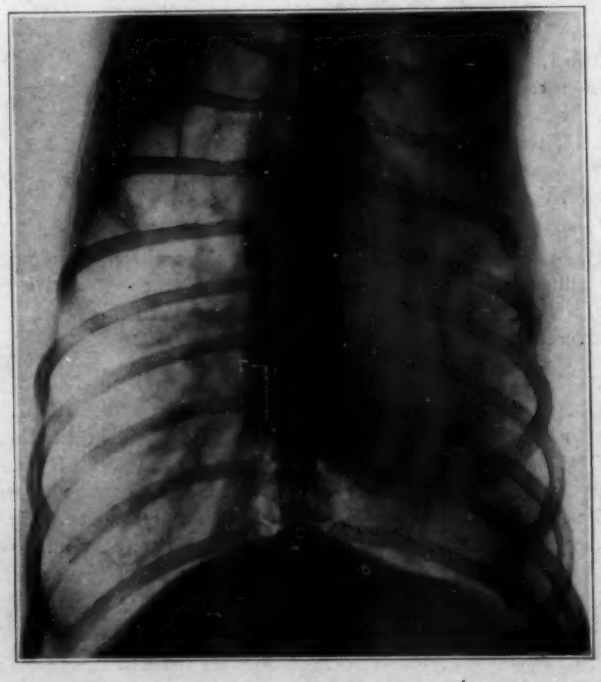

Fig. 41.—Dog Y59, experiment 55, two days after embolism. There is considerable infiltration of the left lower lobe.

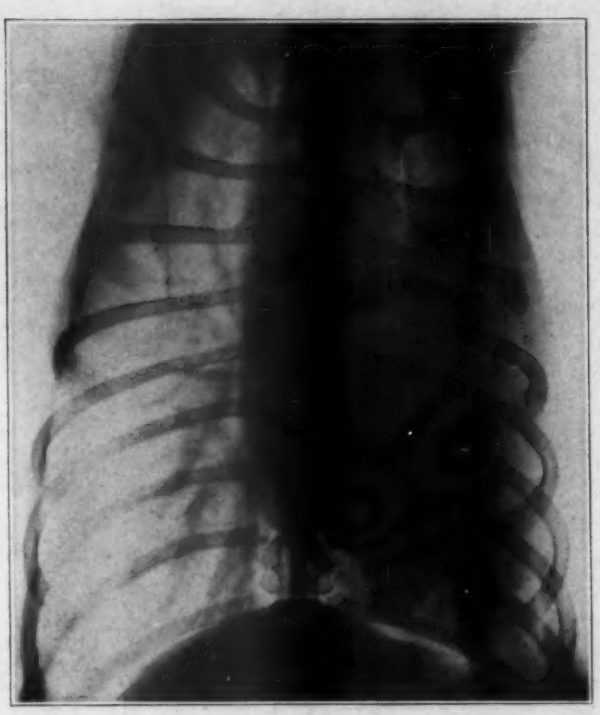

Fig. 42.—Dog Y59, experiment 55, four days after embolism; there is marked consolidation of the left lower lobe.

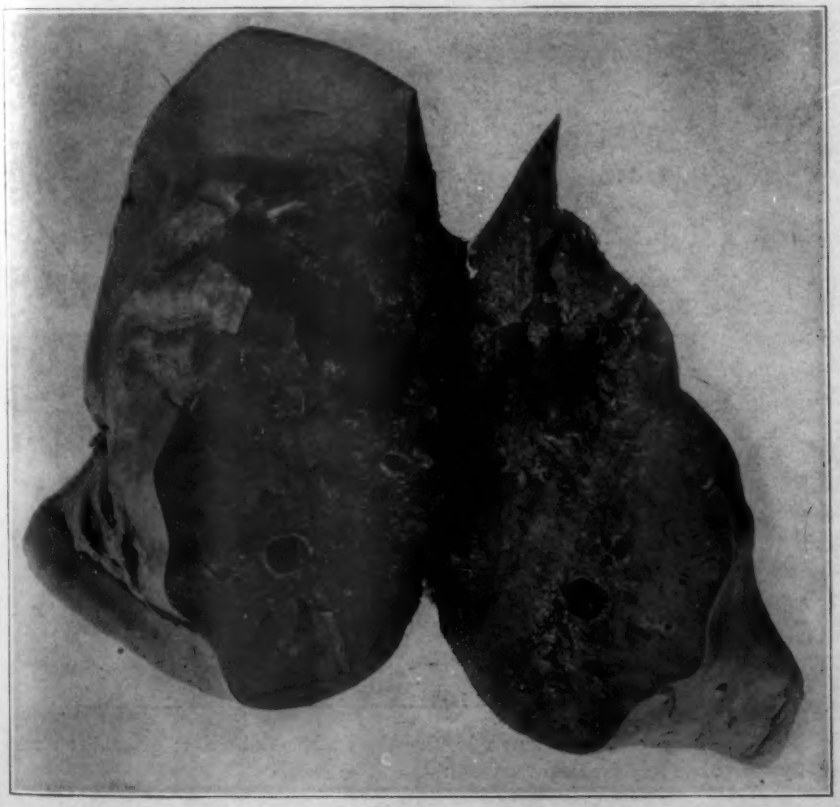

Fig. 43.—Left lower lobe removed from Dog Y59, experiment 55, four days after embolism; a large necrotic walled abscess is present.

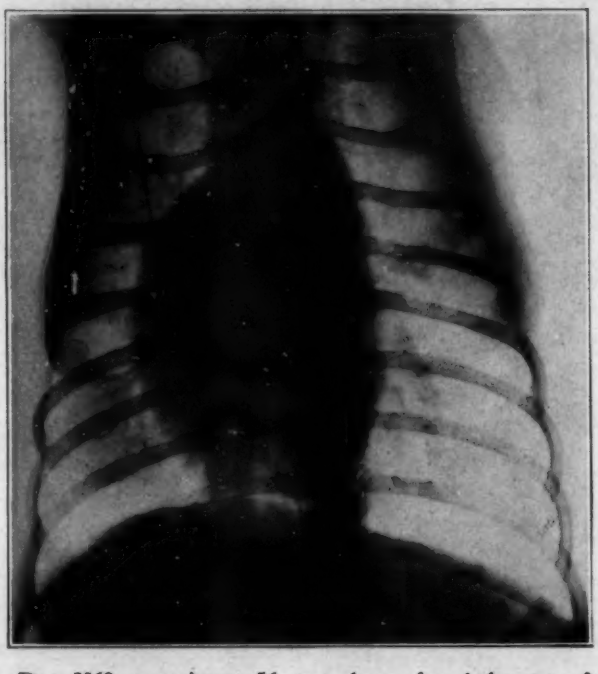

Fig. 44.—Dog Y60, experiment 56, two days after lodgment of an infected embolus in the right middle lobe; there is beginning infiltration.

Fig. 45.—Dog Y60, experiment 56, five days after embolism; a small abscess cavity surrounded by a zone of infiltration can be seen.

March 20, the animal continued ill. The rectal temperature was 40 C. A roentgenogram of the chest showed a dense clouding of the entire lower half of the left lung. The heart was pulled over to the left side (fig. 41).

March 22, the animal remained ill. The rectal temperature was 40 C. A roentgenogram of the chest still showed the same degree of lung consolidation (fig. 42). The entire left lung was removed by operation. There were numerous pleural adhesions and the lung was removed with difficulty. The removed lobe was consolidated and over an area of fluctuation the visceral pleura was markedly thickened. When the lobe was sectioned a large necrotic walled abscess was found (fig. 43).

EXPERIMENT 56.—Dog Y60, weighing 13.8 Kg., March 20, 1926, was given one-fourth grain of morphine. Under procaine anesthesia the femoral vein was

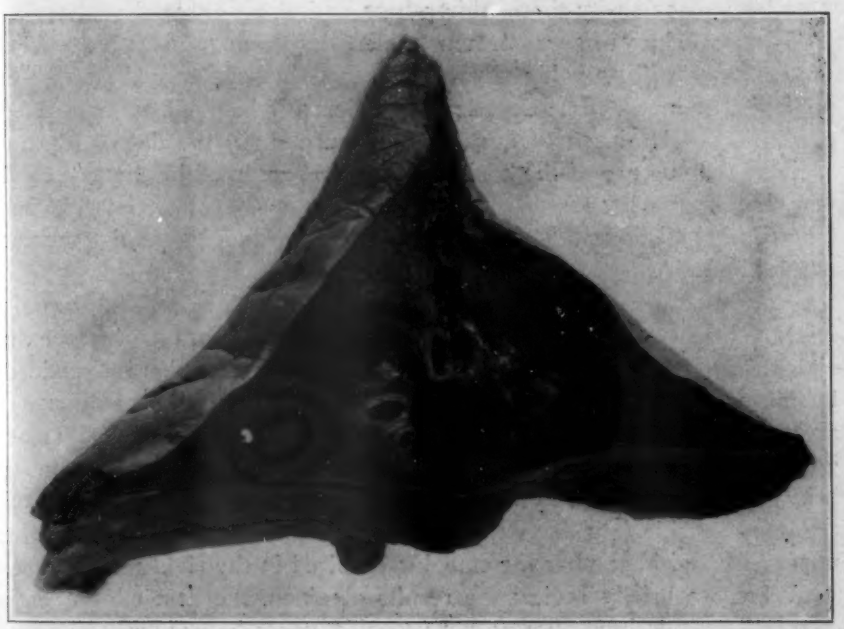

Fig. 46.—Right middle lobe removed from Dog Y60, experiment 56, eight days after embolism; a small abscess cavity is present.

exposed and a portion central to the point of division in the lower leg was removed. The segment of vein was inoculated with cultures of *Bacillus coli* and streptococcus and the prepared embolus was injected into the upper portion of the femoral vein. The vein was ligated and the wound closed with silk sutures. A roentgenogram of the chest localized the foreign body in the left lower lobe.

March 26, six days had elapsed from the date of injection before the embolus localized in the lung. On this date a roentgenogram of the chest revealed the foreign body in the right middle lobe. There was already a slight degree of infiltration about the piece of lead.

March 27, the animal appeared slightly ill but a little food was eaten. A roentgenogram of the chest showed a greater area of density in the right middle lobe (fig. 44). A small cavity appeared within the central area of consolidation.

March 30, the abscess cavity surrounded by a zone of infiltration had increased considerably in size (fig. 45). The abscessed right middle lobe and the remaining lobes of the right lung were removed by operation. On examining the removed lobe, an indurated mass about the size of a walnut could be felt. The visceral pleura overlying the mass was reddened and thickened. When the lobe was sectioned a small abscess cavity was encountered (fig. 46).

COMPARISON AND CONCLUSIONS

From the foregoing protocols it is apparent that we were unable to establish a chronic lung suppuration even in those animals in which the abscess was allowed to remain in the lung for a comparatively long period of time. In Experiments 18, 30 and 32 in which the lesion was examined after thirty-three, thirty-four and thirty-three days, respectively, a healed process was demonstrable. A chronic suppurative process of the lungs will probably never be realized in dogs regardless of the methods employed. The experiences of Scarff [90] help substantiate these conclusions. Drainage of any lung suppuration in the dog is greatly facilitated by nature of the horizontal position assumed by the bronchial tree, but even without such excellent drainage, it is doubtful whether long continued suppuration can be maintained because of the dog's great natural resistive powers.

According to the roentgenograms, the embolus lodged in the left lower lobe in twenty-nine instances and in the right lower lobe in eighteen instances. The reason for such embolus localization is explained by the more direct and straight course pursued by the left pulmonary artery in the dog, and by the characteristic greater volume of blood going to the lower lobes as compared to the upper lobes. In the human being the right pulmonary artery has the advantage over its fellow of the opposite side. In one experiment, the embolus lodged in the right upper lobe and in another the embolus localized in the right middle lobe.

When we briefly review the results obtained, the first experiments demonstrate that lung abscess can probably never be produced in dogs if the infection is introduced by way of the bronchi. The experiments further show that such acute lesions can be produced in dogs if the intravenous route is chosen as the pathway for the transmission of the infective agent to the lung. Successful production is, however, dependent on several factors. Thus, it has been shown that the type of organisms employed is of considerable importance and that certain bacteria must be used in order to produce lung suppuration in the dog. Furthermore, the bacteria must be kept accumulated in a single mass and enclosed in some sort of a capsule to prevent dissemination over a large pulmonary area.

The importance that the physical properties of the embolus hold is strikingly illustrated in the experiments (experiments 16 and 27) in which a covering was not placed about the infected piece of tonsillar

tissue constituting the embolus. In each case a rapidly spreading acute diffuse inflammation developed; a suppurative pneumonitis was produced followed at a later stage by gross necrosis within the affected lung and perforation of the thus formed abscess into the pleural cavity, resulting in death. Similar results were obtained in three experiments in which an infected clot, inoculated with staphylococcus and *Bacillus coli* twenty-four hours previously, was released into the venous circulation. Two of the animals died within forty-eight hours and the third after three days. At necropsy a tremendous infection of the pleura and lung was found. The pathologic process was chiefly limited to the lobe in which the embolus lodged and consisted of an acute diffuse inflammation without gross evidence of tissue destruction.

If the embolus, however, is constructed with a covering, sufficient time is allowed the lung tissues to set up a protective reaction about the point of lodgment of the embolus before the bacteria actually penetrate the lung parenchyma. A well localized inflammatory focus with a peripheral advancing zone of acute inflammation and an increasing central area of necrosis is then gradually permitted to form. Clinically, the embolus consists chiefly of a septic blood clot and in order to conform as nearly as possible with the artificially prepared embolus used in our experiments it must be assumed that the bacteria are in the majority centrally located in a clot that has a somewhat resistant peripheral coat. Should an embolus fail to possess these certain physical properties, a diffuse pneumonitis would be expected such as was produced in the animals in which an infected blood clot was released into the venous circulation. The production of some degree of local tissue immunity made possible by the actual physical make-up of any single infected embolus may play a dominant rôle as to whether lung abscess is or is not to result. Or, we may well hypothecate other methods by which this local immunity is aroused. Thus, in clinical cases one could easily imagine that the organisms to be later transported in abundance in a clot were already being freed in small numbers in the blood stream and thereby setting up a general tissue immunity. Or one could well imagine that smaller, either sterile or infected clots, preceded the major missile and thereby established a specific local tissue immunity in the field in which the larger clot was later to arrive. Certainly, it would appear as if some such problem in tissue immunity was involved.

The fact that abscess occurred in our experiments whether general or local anesthesia was used deserves mention.

IV. PATHOLOGIC STUDIES OF EXPERIMENTALLY PRODUCED LUNG ABSCESS

A pathologic study of lung abscess from the incipient to the final stages following the performance of a uniform procedure aimed at production is obviously desirable. It has been shown that lung abscess

TABLE 5.—*Intravenous Injection Series for Pathologic Studies*

Experiment	Description of Embolus		Location	Duration of Lesion	Pathologic Description of Specimen	
	Material	Organisms			Gross	Microscopic
57 (Y 42)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Left lower lobe	4 hours	Embolus lodged in artery	Beginning leukocytic infiltration of arterial wall
58 (Y 41)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Left lower lobe	8 hours	Embolus lodged in artery with beginning thrombosis	Intima of artery destroyed; considerable leukocytic infiltration of arterial wall; beginning infiltration of lung tissue
59 (Y 45)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Right lower lobe	12 hours	Thrombosis of vessels with beginning hemorrhagic infiltration	Coats of artery densely infiltrated; considerable leukocytic infiltration of lung tissue
60 (Y 43)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Right lower lobe	18 hours	Thrombosis and slight infiltration about vessel	Cellular exudate in lumen of vessel; vessel coats difficult to distinguish; early acute inflammation of lung tissue
61 (Y 44)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Right lower lobe	24 hours	Thrombosis; infiltration more marked	Vessel wall practically destroyed; marked acute inflammation of lung tissue
62 (Y 46)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Left lower lobe	36 hours	Thrombosis; considerable surrounding infiltration	Arterial wall destroyed; pus cells and tissue débris present; surrounding lung tissue, including bronchi, showed acute inflammation
63 (Y 47)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Left lower lobe	2 days	Infiltration marked; beginning area of necrosis in center	Necrotic walled abscess present; surrounding inflammation marked
64 (Y 49)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Left lower lobe	3 days	Large area of infiltration containing small necrotic walled abscess	Extensive acute inflammation
65 (Y 50)	Vein, lead, blood	Pneumococcus, B. coli, streptococcus, Staphylococcus aureus	Right lower lobe	4 days	Same as experiment 64; abscess larger	Marked and extensive acute inflammation

can be produced by the placement of infected materials into the venous system, and that the lesion is sure to occur with the employment of certain types of organisms in the embolus. With such knowledge at our disposal, examination of similarly produced lesions at various periods of time is possible.

Method of Collecting Specimens.—In a series of nine experiments (table 5), a segment of femoral vein was prepared in the usual manner, inoculated with cultures of *Staphylococcus aureus,* pneumococcus, *Bacillus coli* and streptococcus and injected into the jugular vein. Roentgenograms of the chest were made immediately after the injection in order to determine the position of the embolus. The specimens were obtained either by sacrificing the animals or by performing a lobectomy,[92] and the lesions were examined grossly and microscopically after four, eight, twelve, eighteen, twenty-four, thirty-six, forty-eight, seventy-two and ninety-six hours. The lung lobe specimen was injected intrabronchially with formalin (10 per cent) and allowed to remain in the solution for a few days. The lobe was thus brought to the desired normal dimensions present during life.

DESCRIPTION OF SPECIMENS

Four Hour Specimen.—Left Lower Lobe: The surface of the lung appeared normal. A section taken through the lobe in a plane perpendicular to the main bronchus and artery bisecting the embolus showed blood clot formation within the lumen of the artery. Distally, no infarction was demonstrable. A section for microscopic study was taken transversely through the artery. Histologically, a slight degree of hemorrhage could be seen within the arterial wall and a few leukocytes infiltrated the muscular coat. The intima of the artery was fairly intact throughout. A few leukocytes could be seen in the adjoining lung tissue. The picture was that of a beginning inflammation.

Eight Hour Specimen.—Left Lower Lobe: The surface of the lung appeared normal. A section through the lobe in a plane perpendicular to the main bronchus and artery, and at a point midway between the hilum and the tip of the lobe, bisected the embolus plugging the lumen of the artery. A bronchus lay in close proximity. The embolus and surrounding blood clot was already quite adherent to the vessel wall. The thrombus extended distally for about 2 cm. No infarction was demonstrable. A section for microscopic study was taken through the central area of thrombosis and bisecting the embolus. Histologically, the vein, constituting the outer wall of the embolus, could be seen within the lumen of the artery. The intima of the artery was destroyed. There was considerable infiltration of the arterial wall. A few cells could be seen in the surrounding lung tissue. The cells were chiefly leukocytes.

92. We were led to consider the possibility of operative removal of an abscessed lung because of a desire to preserve the life of the animal. A highly satisfactory technic was evolved, the results of which have been published separately (Schlueter, S. A., and Weidlein, I. F.: Surgery of the Lung: Experimental Lobectomy and Pneumectomy, Arch. Surg. **13**:459 [Oct.] 1926).

Twelve Hour Specimen.—Right Lower Lobe: The surface of the lobe appeared normal. A section taken in a plane perpendicular to the bronchus and artery revealed the embolus lodged in an arterial lumen at a point approximately two thirds of the distance from the hilum to the tip of the lobe. The artery lay near a large bronchus. Induration could be felt in the tissues surrounding the vessel and a narrow peripheral zone of hemorrhagic infiltration was visible. The embolus and adjoining thrombus were adherent to the vessel wall. Thrombosis extended distally for a considerable distance but there was no infarction. A section for microscopic examination was taken through and about the area of the embolus. Histologically, all coats of the arterial wall were densely infiltrated with leukocytes. The surrounding lung tissue also showed considerable leukocytic infiltration. Numerous areas of fresh hemorrhage were present. The walls of an adjoining bronchus showed leukocytic infiltration. Some cellular exudate was present in the bronchial lumen.

Eighteen Hour Specimen.—Right Lower Lobe: The surface of the lung appeared normal. Section taken through the lobe in a plane perpendicular to the direction of the bronchus and artery revealed the lead and bits of silk within the lumen of the artery at a point about midway between the hilum and the tip of the lobe. There was gross evidence of infiltration and edema in a small area surrounding the point of lodgment of the embolus. Thrombosis extended distally along the lumen of the vessel for a considerable distance. A section for microscopic examination was taken through the area in which the embolus was lodged. Histologically, the lumen of the artery was entirely destroyed. Fibrin, cellular exudate and tissue débris filled the former vessel lumen. The vessel's coats were difficult to distinguish because of the dense infiltration with leukocytes. The inflammatory exudate extended far into the surrounding lung tissue. Numerous areas of fresh hemorrhage were present. An adjoining bronchus was included in the area of inflammation. Histologically, the picture was already that of a true abscess.

Twenty-Four Hour Specimen.—Right Lower Lobe: The surface of the lobe appeared normal. A section taken through the lobe in a plane perpendicular to the axis of the bronchus and artery revealed the piece of lead and bits of silk lying within the lumen of the artery about midway between the hilum and the tip of the lobe. Definite thickening could be felt in the tissues surrounding this area. Gross evidence of infiltration and hemorrhage was more marked. A section for microscopic examination was taken through this area. Histologically, bits of silk were seen within the arterial lumen together with considerable cellular exudate and tissue débris. The walls of the artery were still more difficult to distinguish because of the dense infiltration with leukocytes. The adjoining lung tissue was the seat of even more marked inflammation.

Thirty-Six Hour Specimen.—Left Lower Lobe: The surface of the lobe appeared normal. An area of density could be felt within lying about midway between the hilum and the tip of the lobe. A section taken through this area traversed the point of lodgment of the embolus. Hemorrhagic infiltration extended radially from this cavity, in which the piece of lead was recovered, for a distance of about 0.5 cm. The vessel wall appeared necrotic. Thrombosis extended distally for a considerable distance. Histologically, the arterial wall was almost completely destroyed. Pus cells and tissue débris occupied the lumen. The abscess wall consisted of leukocytes. Leukocytic infiltration and fresh hemorrhage extended into the surrounding lung tissue. An adjoining bronchus showed destruction of its epithelial lining and in some parts a considerable portion of its entire wall.

Forty-Eight Hour Specimen.—Left Lower Lobe: The surface of the lung appeared normal. A small firm nodule could be felt within. A section taken through this area revealed bits of silk and the piece of lead lying within a necrotic arterial wall. Hemorrhagic infiltration extended radially for about 0.5 cm. Distal to this point, the vessel was not thrombosed. Histologically, a small necrotic walled abscess was present. The wall of the abscess consisted of a dense zone of leukocytes. The leukocytic infiltration extended for a great distance into the surrounding lung tissue. Numerous areas of fresh hemorrhage were present throughout. An adjoining bronchial wall was partially destroyed.

Seventy-Two Hour Specimen.—Left Lower Lobe: A small mass could be felt within the lobe. The visceral pleura overlying this area was thickened. A section taken through the mass passed through a small abscess cavity about 1 cm. in diameter containing the piece of lead and bits of silk. Necrotic bits of membranous tissue, in all probability the remnants of the vein used as the embolus, were also present. The walls of the artery going to the lobe could be identified lying centrally to the abscess and the vessel on both sides of the abscess cavity was thrombosed. On all sides of the abscess there was a zone of hemorrhagic infiltration approximately 1 cm. in width. The portion of the lung distal to the abscess was quite firm, even to the tip of the lobe. A section for microscopic study was taken through the wall of the abscess. Histologically, the inner wall of the abscess consisted of broken down cells and leukocytes densely packed together. Extensive acute inflammation existed in the surrounding lung tissue.

Ninety-Six Hour Specimen.—Right Lower Lobe: The lower half of the lobe was quite firmly consolidated and dark red. The visceral pleura was thickened and covered with a fibrinous exudate. A section taken through the area of consolidation revealed a small abscess cavity from approximately 1 to 2 cm. in diameter, containing bits of silk, the lead filing and necrotic remnants of vein wall. Thrombosis of the artery extended centrally from the abscess cavity and hemorrhagic infiltration was present for a considerable distance about the artery. The lung tissue surrounding the abscess was densely consolidated. A section for microscopic study was taken through the wall of the abscess. Histologically, the section showed a picture similar to the previous one except for a more marked and more widespread acute inflammation in the surrounding lung tissue. The walls of the bronchus lying near the abscess cavity were completely destroyed.

SUMMARY

Histologically, it is apparent that a true abscess is already present as early as the eighteenth or twenty-fourth hour after the lodgment of the infected embolus, and that the lesion is definitely formed after thirty-six hours. In the thirty-six hour specimen, the wall of a small bronchus is almost entirely destroyed. The extensive surrounding pneumonitis seen in the three and four day specimens simulates the lung infiltration of the early stages. The difference is one of degree only.

The entire pathologic process of embolic lung abscess can be depicted as follows: Following the lodgment of an infected embolus in a pulmonary artery, thrombosis of the vessel occurs. A cellular exudate forms in the vessel wall. The latter is gradually destroyed by bacterial action and the adjoining lung tissue becomes involved in the inflammatory

process. Liquefaction of tissue within the inflammatory focus occurs. More and more parenchymatous structures, including bronchial elements, are gradually absorbed. The abscess increases in size and an attempt to halt the destructive forces is made by the production of a surrounding wall of fibrous tissue. If the infection is overcome, healing takes place by scar tissue formation. The rapidity of resolution is dependent on the virulence of the organisms, whether or not evacuation into a bronchus occurs, and on the strength of the resistive powers.

The material dealt with in our study concerns only the extrabronchial or parenchymatous type of lung abscess, and the histologic evidence that a moderately sized bronchus can be destroyed in the early stages of a true abscess has been presented. This would seem to indicate the difficulty of determining from the examination of a chronic lesion whether the abscess was originally an extrabronchial or bronchiectatic lung abscess. In our opinion, the site of the initial infectious process cannot be established by the study of the lesion in a late stage. Histologically, fibrous tissue lined the abscess cavities in the lesions that were permitted to enter into a healing stage. It has been shown [1] that in clinical cases bronchial epithelium at times lines an abscess cavity, but we believe that this process of epithelialization occurs as a later event in the lesions which have become chronic and which communicate with a bronchus. We are unable to furnish experimental evidence, however, which would demonstrate the possibility of an epithelial proliferation from a bronchus into a long standing parenchymatous lung abscess because of our inability to produce a chronic lung abscess experimentally.

V. GENERAL COMMENT AND CONCLUSIONS

Pathologically, lung abscess consist of two types, bronchiectatic abscess and extrabronchial or parenchymatous abscess. The initial anatomic position assumed by the infecting organisms determines the type that is to follow. Thus the first type originates within the air passages, while the second type begins within the parenchymatous tissues. Each type is entirely dependent on a separate and distinct mechanism by which bacteria are brought to the initial site for implantation. In the parenchymatous abscess this mechanism consists in blood stream transmission of bacteria to the lung while in the bronchiectatic abscess infection is introduced by way of the air passages.

An attempt is made to show that the type of lung abscess developing as a sequel to operation possesses certain distinctive clinical characteristics which when correctly interpreted and defined in terms of a pathologic process place this lung abscess in the true or parenchymatous class, with which no other source of infection than a hematogenous one can be associated.

Our belief that postoperative lung abscess results from embolism, a mechanism produced by the dislodgment of an infected thrombus from the vessels of the operative area, is based on the following facts:

1. The definitely proved existence of the condition of fatal postoperative pulmonary embolism. This supposes the possible scattering from any wound of single or multiple emboli into the venous circulation.

2. The frequent development of lung abscess after operations performed in infected or potentially infected fields. In this class we refer particularly to nose and throat operations, especially tonsillectomy, and to operations performed on the gastro-intestinal tract.

3. The high percentage of occurrence after operations performed in mobile operative areas. Thrombi are easily dislodged from such regions as the pharynx and epigastrium. In operations on the brain in which the skull acts as a splint the percentage of postoperative pulmonary complications is almost nil.

4. The not uncommon appearance after operations in which local anesthesia is employed.

5. The failure to prevent postoperative pulmonary complications with the constantly improved methods of giving inhalation anesthesia.

6. The greater frequency of lower lobe involvement. This is explained by the greater volume of blood and the more direct course of the pulmonary artery to these lobes.

7. The often symptom free period following the operation before the onset of the complication. If the aspiration mechanism were the causative factor, the appearance of the symptoms would be early.

8. The sudden pain in the chest that frequently constitutes the initial symptom and the often severe and stormy associated clinical course that follows before rupture and evacuation occur.

9. The acknowledgment by bronchoscopists that typical lung abscess is rare with the lodgment of foreign bodies even deep in the air passages.

10. The unsuccessful attempts at experimental production in animals by the introduction of infected materials by way of the air passages, either by transtracheal implantation or by aspiration.

11. The comparative ease with which lung abscess can be produced by the intravenous injection of infected materials.

A method is described for the production of lung abscess by freeing infected emboli into the venous circuit. Success depends on the type of organism employed and on the physical property of the embolus. The latter will permit the establishment of some degree of local tissue immunity so that the lesion remains localized and thereby assists toward the prevention of a rapidly spreading acute diffuse inflammation.

The comparatively early formation of a lung abscess, once lodgment of an infected embolus has occurred, and the rapid progress of the destructive lesion illustrates why the condition usually produces such grave clinical symptoms.

A reduction in the number of postoperative lung abscess cases is possible, but the condition can probably never be entirely eradicated. The best prophylactic measures consist of (1) the prevention of infection in the operative field whenever possible; (2) the reduction of operative trauma to a minimum, and (3) the avoidance of mass ligation of tissues.

GENERALIZED OSSEOUS METASTASES SECONDARY TO ATROPHIC SCIRRHOUS CARCINOMA OF LEFT BREAST *

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Second only to etiology, the most important phase of the practical study of tumors is the determination of the anatomic and biologic factors that facilitate or prevent metastases. The cause of cancer is as yet unsolved, but we know that the disease, regardless of its site of origin, ultimately spreads as metastatic growths throughout the body. The metastases may occur late or early, in nearby tissues or in more remote organs, and they are the important determining factor in cancer—usually bringing about the fatal result. Metastases represent cancer cells that have traveled to the point found and are growing as secondary tumors. Whether these detached tumor cells find their way by "spontaneous movements," by "permeation" or by "forcible dissemination," as has been stated by various writers, is of no particular importance.

There are two general routes of tumor cell dissemination, lymphatic and blood. In most instances cancer metastases occur by the lymphatic route, but there can be no doubt that cancer cells obtain access to the blood by invasion of the small veins in the vicinity of the primary tumor and even of the secondary growths. Paget [1] stated that cancer cell embolism is an impartial process to which all organs are liable. He further believed that in cancer of the breast "the bones suffer in a special way which cannot be explained by any theory of embolism alone." The observation of cancer metastases in the various organs does not show an impartial distribution, or at least evidence of equal involvement of all organs on an impartial basis is not found. Some organs are almost regularly invaded, others rarely. We must assume that certain organs act as friendly ports for the entrance and selective harbors for the growth of emboli that lodge in them, while in other organs the lodged emboli are destroyed or prevented from growing. Handley [2] stated:

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1. Paget, Stephens: The Distribution of Secondary Growths in Cancer of the Breast, Lancet **1**:571, 1889.

2. Handley, W. S.: Cancer of the Breast, London, John Murray, 1906, pp. 32, 36 and 37.

Cancer cells excite thrombosis and the thrombus as it organizes and contracts destroys them. Cancer in lymphatic vessels, as far as I have seen, excites no such thrombosis, a fact to which Schmidt draws attention. Hence, there is no doubt that although carcinoma often attains access to the blood almost as early as to the lymph, its dissemination takes place almost entirely by the lymphatic and not by the blood vessels.

The practically constant physiologic hyperemia of the bone marrow and the slowing of the blood current within the dilated capillaries, with the adherence of the tumor cells to the endothelium, have been explained [3] as favorable factors for the localization and growth of embolic tumor cells.

DISTRIBUTION AND FREQUENCY OF OSSEOUS METASTASES FROM MAMMARY CARCINOMA

Studies of bone metastases have been confined to three fields: First, those associated with spontaneous fractures; second, those found at necropsy, and, third, those observed by roentgen-ray examination. Statistics from these are of little value because of their incompleteness and the inexactitude of the records. Necropsies, which are the most valuable means for such study, rarely extend to a critical examination of the osseous system. Until such structures are universally examined as searchingly as is now done with the lungs and liver, for instance, little will be known as to facts of osseous metastases.

Gross,[4] in 423 compiled necropsies, found secondary tumors in the brain in forty cases, or 9.4 per cent; in the pleura in 178 cases, or 42 per cent; in the liver in 206 cases, or 48.6 per cent, and in the bones in eighty-seven cases, or 20.5 per cent. Paget,[1] in 650 necropsy records of breast cases, found the cranial bones involved thirty-six times, the femur eighteen times and the humerus ten times. Williams,[5] in forty-four necropsies, found the femur affected four times (each once and both twice), the humerus twice (each once), the vertebrae, the tibia and ribs each once. In 893 compiled necropsies, he reported bone metastases in 26.5 per cent of the cases. The percentage site-incidence of these metastases were: cranium, 24; vertebrae, 19.2; ribs, 19.1; femur, 14.5; humerus, 9.3; innominate, 6.5; clavicle, 4.2; scapula, 1.2; leg bones, 0.9; forearm bones, 0.45, and sternum, 0.45. Direct extension of the primary tumor to the ribs, sternum, clavicle and upper end of the humerus was not included in this series.

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3. Knox, Leila C.: Relationship of Massage to Metastasis in Malignant Tumors, Ann. Surg. **75**:129-142 (Feb.) 1922.

4. Gross, cited by Mann, M. D.: American System of Gynecology, Philadelphia, Lea Brothers & Co., 1888, vol. 2, p. 297.

5. Williams, W. R.: The Natural History of Cancer, New York, William Wood & Co., 1908, p. 431.

The nearer a bone is to the site of a primary cancer, the greater is its liability to secondary involvement. In mammary cancers, the ribs and sternum are most frequently involved. The cranial bones, spine, femur and humerus are, in the order given, the next most common seats for metastatic growths. It is generally stated that the clavicle is rarely involved in breast cancers, thus being an exception to the general rule of proximity.

Fig. 1.—Metastatic growth of left superior maxilla involving rim of orbit; metastasis with necrosis in frontal bone.

Carcinoma invades the bones distal to the knee and elbow only in the rarest instances. The absence of records of spontaneous fracture in the distal bones does not prove that their escape from cancerous invasion is real. Invasion may be there, but we fail to search for it; and the demand on these parts is not so severe as on the other bones where spontaneous fractures are seen. We do not agree with Handley,[2] who says that these

bones escape simply because the patient invariably dies before growth has spread along the deep fascia far enough to reach them.

As the result of his work on cancer of the breast, Handley [2] regarded hematogenic embolism as playing an unimportant part in the formation of metastases, and explained all parietal and most visceral metastases as

Fig. 2.—Metastases in occipital, left parietal, vertebral and innominate bones; occipital protuberances destroyed.

being permeations along lymph spaces and fascial planes. Permeation plays an important part in the spread of cancer, especially cancer of the breast; embolism, however, can by no means be disregarded and is, in fact, the only adequate explanation in some cases as in the one reported here.

REPORT OF CASE

History.—Mrs. L. W., white, aged 56, a widow, entered St. Mary's Hospital, Jefferson City, Mo., July 27, 1925, for fracture of the left thigh. No family history was obtained and the personal history, until the present illness, was unimportant. She had had the usual childhood diseases. The menses had been normal

Fig. 3.—Calvarium: circular area in midline of frontal bone made for injection of brain.

with cessation at the age of 41. She married at the age of 15 and gave birth to her only child six years later. There was no history of miscarriages, operations or venereal diseases, by name or symptom.

There was spontaneous fracture of the right humerus, June 22, 1925, as the patient jumped out of bed when awakened by a rat between her legs. She landed on her feet but did not strike her arm. This arm, carried in splints, gave little

trouble. For weeks she had difficulty in walking, and had found it necessary to depend on support. The day prior to admission to the hospital, in crossing the room supported by a chair which slipped, she felt something "crack" in her thigh, and she fell.

Physical Examination and Treatment.—Examination on admission revealed a recent fracture of the left femur, an unhealed fracture of the right humerus, a nodular swelling in the left breast, enlarged left axillary lymph nodes and cystitis. The patient was emaciated and covered with decubitus ulcers; she was of poor intellect and made irrational statements and answers. The blood Wassermann reaction was negative. The blood counts were: erythrocytes, 4,320,000; hemoglobin, 85 per cent; leukocytes, 10,700; neutrophils, 70 per cent; lymphocytes, 15 per cent, and transitionals, 15 per cent. A diagnosis of generalized tuberculosis with bone involvement was made.

The patient remained in the hospital with her extremity in splints and during her stay suffered continuously with pain, especially marked about the frontal region of the head, spine, hips and extremities. Morphine was given regularly but with little relief. During October, the left lower extremity became swollen, and near the end of the month the foot and leg became gangrenous. On November 5, the left leg was amputated at the middle of the thigh. The part amputated was not submitted to pathologic examination. The patient died, Dec. 10, 1925.

Necropsy.—The body, that of a well developed, but poorly nourished white woman of 55 to 60 years of age, had been embalmed, and the palpable tissues were fairly firm in consistency. There was a fracture of the right arm at the junction of the lower and middle thirds. Below the point of crepitation was an elliptical firm growth involving the shaft of the bone. There was an angulation with the lower fragment being sharply bent inward. Immediately above the left elbow was a fracture of the left arm. Below the point of crepitation was an irregular, hard mass involving the lower fragment and extending to the elbow. At the junction of the upper two fifths with the lower three fifths of the right thigh was an accidental postmortem fracture. The lower half of the left thigh, leg and foot were absent. At the line of amputation was a recent ellipictal, unhealed surgical incision partially closed by sutures. From the edges of the wound exuded a thick, yellow foul smelling pus.

The tissues of the right leg, ankle and foot were edematous. In the posterior thoracic region from the sixth to the eleventh vertebrae, inclusive, the skin, subcutaneous tissues and a part of the muscle tissue were necrosed away, and in this reddish-black area, muscles, ligaments and some of the spinous processes were seen. An oval area covering the prominences of the sacrum was involved in a similar necrotic process. The skin and tissues of the outer surface of the right ankle and side of the foot were necrotic for an area 3.5 by 5 cm. This area was reddish black, and its surface was bathed by a thick, putrid pus. This exudate extended between the bones of the foot, covering their articular surfaces.

Both pleural cavities were largely obliterated by firm white bands of adhesions. The lungs had irregular white, opaque, retracted scars at their apexes. Surrounding these were multiple 1 to 2 mm. firm white tubercles. The heart was of normal size; its chambers contained soft, loose cruor clots. The aorta, the coronary, splenic, renal and pancreatic arteries were thickened, and contained opaque yellow plaques of arteriosclerosis, some of which were calcified.

The spleen, pancreas, liver, gallbladder, bile ducts, suprarenals and the gastrointestinal tract showed no appreciable gross lesions.

The kidneys were normal in size. In the cortex were multiple bilateral yellow to gray abscesses. The tubules appeared as grayish to yellow streaks running to the

pelves, which were dilated and filled with grayish-yellow pus. The mucosa of the urinary bladder was covered by a necrotic, gumatous, foul smelling, grayish to black exudate. The lining, containing multiple petechial hemorrhages, was gray to black.

The ovaries, small and firm, contained multiple white scars. The uterus was small and firm, the canal partially obliterated and the cervix closed by a plug of mucus.

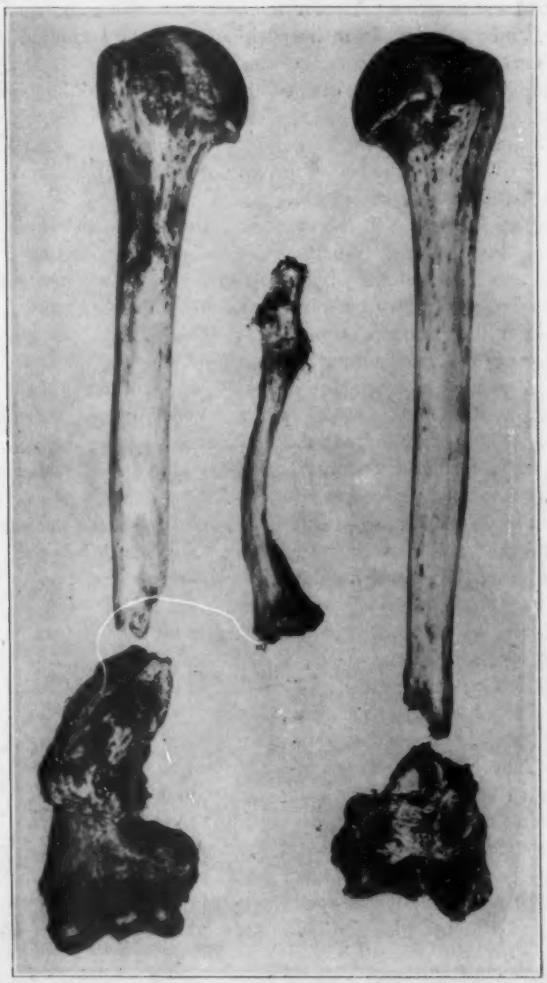

Fig. 4.—Areas of tumor invasion with resorption of bone in humeri and left clavicle, with fractures of humeri.

The meninges had adhesions to the inner table of the skull along the superior longitudinal sinus. The brain was diffluent.

The left breast had an atrophic, retracted nipple lacking in pigment, overlying an elliptical, dense, firm tumor involving the organ for an area 5.8 by 8.2 cm. The atrophic breast, with its contained tumor, was firmly adherent to the skin covering it, to the deeper tissues and to the chest wall. The skin over the breast

was dry, scaly, contracted, devoid of folds and roughened; in zones it was free from pigment, but in other portions it was a dirty, brownish gray. There were two firm nodules at the lower and inner margin of the organ, one measuring 1 cm. and the other 1.4 cm. in diameter. These were fixed to deeper structures and to the smooth, stretched, bluish skin that covered them. On section the surface 1 cm. distant from the nipple revealed a firm, grayish-white tumor mass fusing with the deeper layers of the skin and extending for a depth of 3 cm. to involve the gland proper and the muscles posterior to the breast. The margins of the growth were irregular, and radiating from its periphery into surrounding structures were grayish white crab-leg-like process.

The right breast was twice the size of the left, was pendulous and contained no palpable nodules.

Aside from the superficial metastases at the margin of the left breast there was an elevated, oval, firm, grayish white, immovable, subcutaneous tumor 3 by 2 cm. lying immediately above the right zygomatic arch.

Of the lymphoid tissue, the left axillary, supraclavicular and infraclavicular nodes as groups were enlarged, firm and fixed to surrounding structures. On section the surfaces were almost uniformly grayish white with only small islands of recognizable lymphoid structure present. The tracheobronchial nodes were enlarged and firm but uniformly black. Neither these nor the mesenteric or retroperitoneal groups showed tumor metastases.

Osseous System: The superior maxilla had an irregular grayish white tumor growth involving the nasal process, the orbital surface, the facial surface and the malar process, from the articulation of the nasal process with the frontal bone to the maxillary process of the malar bone, and from the orbital surface downward toward the canine process for 1.8 cm. In the central portion of the growth on the facial surface of the bone was the infra-orbital foramen with the infra-orbital nerve and artery surrounded by tumor growth. The growth extended to the malar bone and involved its orbital process, and to a slight extent the articular margin of its maxillary process.

The other bones of the skull contained tumor metastases with areas of consequent bone necrosis as follows:

In the frontal bone above the right frontal eminence, an oval area, 3.2 by 2.8 cm., with irregular edges, extended through the skull.

In the right parietal bone there were three oval areas. The first, 1.1 cm. in diameter, was 3.2 cm. posterior to the frontoparietal suture and 2 cm. to the right of the interparietal suture. Posterior to this almost in a direct line, 2.5 cm. and 6 cm., respectively, were the other two, measuring 1.2 cm. and 1.7 cm. each.

In the left parietal bone, posterior to the parietal prominence and extending backward to the parietal-occipital suture, was an irregular motheaten-like area, 5.2 by 4.4 cm. In portions of this area there was complete necrosis of both tables of bone. Around the margin small islands and projections of cancellous bone remained.

In the occipital bone, an area 3.2 by 2.3 cm. involved the entire thickness of the skull, centering about the external and internal occipital protuberances.

The right fourth rib, beginning at a point 9 cm. from its head and extending to the costal cartilage, was white like dead bone, and its medulla was filled with a firm, white tumor growth. The left second and third and the right third and fourth ribs were fractured within 2 cm. of the costochondral line at points where there were grayish white tumor growths with bone necrosis.

In the vertebral column there was a moderate kyphosis and scoliosis involving the junction of the thoracic and lumbar vertebrae. The bodies of the twelfth dorsal and the first lumbar vertebrae were almost completely destroyed, allowing a

collapse of the spine at this area with the thoracic section being bent forward at an angle of 25 degrees. Areas of tumor growth with bone necrosis were present in the anterior portion of the bodies of the third and fourth lumbar vertebrae.

The left clavicle had an area of grayish white tumor growth with bone destruction involving the acromial end, destroying in a semilunar area one third of

Fig. 5.—Ancient fracture of left femur with excessive callus containing tumor tissue, and erosion of surgical neck with incomplete fracture; multiple metastases in right femur, fibula and tibia, with unseparated fracture near lower end of tibia.

the articular surface and an area 1.5 cm. in diameter internal to the oblique line on the under surface, and extending to the deltoid tubercle. The right clavicle, 3.8 cm. from the acromial end, was the seat of an ancient, healed fracture with scant callus formation.

The left humerus had a recent transverse fracture 4.5 cm. above the articular surface of its lower end. The bone of the lower fragment to the articular car-

tilage was increased in size by an uneven elliptical callus in which there was extensive tumor tissue growth. The canal of the upper fragment of bone was filled for some distance by white, fairly firm tumor tissue. Near the middle of the shaft on the posterior surface was an irregular, motheaten area of bone necrosis, 1.2 by 1 cm., with tumor growth filling the space.

The right humerus, 11 cm. above the articular surface of the lower extremity, had a recent fracture with callus formation on all surfaces. The bone at this location was thin, and the canal filled with grayish white tumor tissue. The lower fragment was nodular, uneven and bent inward with a sharp angulation. There was a previous fracture 4 cm. below this where the angulation was more pronounced, and the callus, consisting in part of tumor tissue, was excessive. Beginning at the surgical neck on the external surface at the lower extremity of the bicipital groove and extending for 5.5 cm. downward, and from the inner lip of the bicipital groove backward 4.3 cm., involving the external surface, and on to the posterior surface was a tumor growth with bone necrosis involving three fifths of the bone circumference, or all but a portion 2 cm. wide.

The pelvic bones contained multiple tumor growths with corresponding areas of bone necrosis at the following sites:

At their articulation the left os pubis and left ileum had an area 4.6 by 3.6 cm., one fourth of which involved the terminal part of the upper surface of the os pubis; the remaining portion extended to the internal iliac fossa, laterally from the linea ileo pectinea to the upper rim of the acetabulum. Posterior to this 4.5 cm. a 1 cm. area involved both surfaces of the ileum. In front of the posterior superior spinous process and below the crest on the external surface, an area 1.6 by 1.7 cm. and another 4.6 by 2.3 cm. destroyed parts of the posterior superior and inferior spinous process.

The right iliac bone had along its crest 3.5 cm. posterior to the anterior superior spinous process a half moon area with the flat side 4 cm. along the crest and the circular edge 1.6 cm. on to the inner surface of the bone. Immediately above the posterior superior spinous process, separated from the crest by a 4 mm. rim of bone, involving the inner and outer surfaces, more markedly the latter, centered on the external table opposite the articulation between this bone and the sacrum was a circular area 4.5 cm. in diameter, and in front of the posterior interspinous notch was another measuring 9 mm.

On the posterior surface of the right ischium an area involved the greater portion of the inner surface and one half the external surface of the body, from the upper margin of the bone but not damaging the acetabulum, down and on to the internal lip of the tuberosity, and invading on the internal surface the first 1 cm. of the ascending ramus.

In the right os pubis on the external surface of the descending ramus at the line of fusion with the ascending ramus was an area 2.6 cm. which invaded the bone medulla but not the inner surface.

The left femur was the site of a recent amputation at the middle of the shaft. A recent incomplete fracture of the neck almost separated the head of the bone from its shaft. Approximately one half of the neck and of the shaft between the spiral line and the lesser trochanter had been eroded and replaced by firm, grayish-white tumor tissue. The involvement extended from the superior border of the neck downward 5 cm. to a point on the anterior surface of the femur corresponding to the lower border of the lesser trochanter; transversely at the widest place it measured 3.4 cm. A relatively recent transverse fracture of the shaft was present 8.5 cm. below the lower border of the lesser trochanter. The upper fragment was displaced outward and forward while the lower was outward, backward and upward. The ends of the bone, which were separated by a

distance of 6.5 cm., and the intervening space were encompassed by a heavy, firm callus consisting in part of tumor growth and spicules of calcified bone. The anterior surface of the upper fragment at the lower end was the seat of tumor growth and bone resorption, 2.2 cm. wide, for the distal 3 cm. Involving the linea aspera, 4.5 cm. below the lower margin of the greater trochanter, was an oval 6 mm. area of tumor growth and bone necrosis.

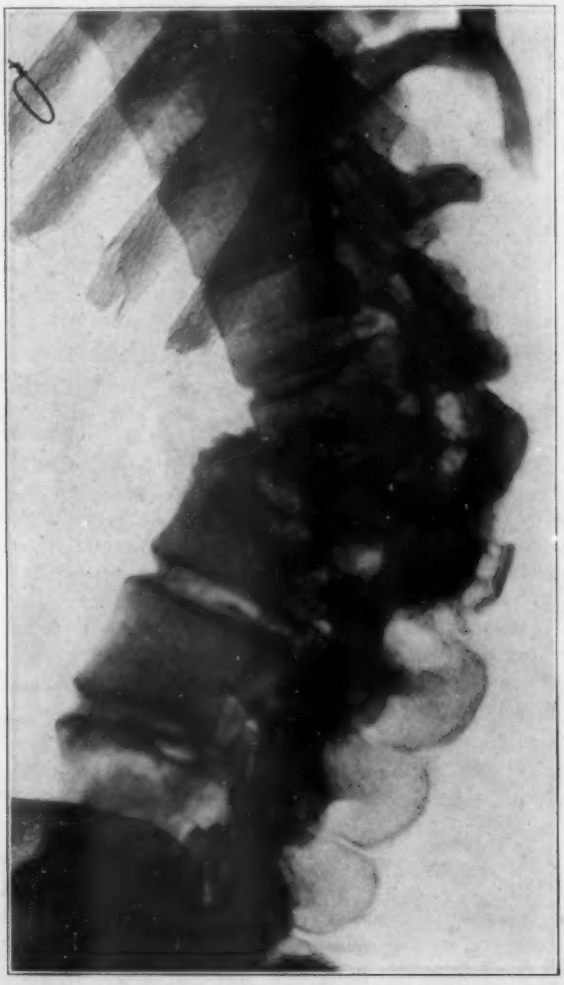

Fig. 6.—Spine showing collapse of twelfth dorsal and first lumbar vertebrae; tumor metastases with rarefaction of anterior portion of the bodies of the third and fourth lumbar vertebrae.

The right femur at the upper portion of the shaft involving the greater trochanter had an area of tumor growth with marked bone necrosis on the anterior and external surfaces extending from above downward for 5 cm. and involving three sevenths of the bone circumference. The cancellous bone was completely resorbed and replaced by a grayish-white tumor. The shaft 7.3 cm. below the

lesser trochanter was the seat of a fracture accidentally made in removing the body from the shipping container. At this location the bone was markedly thinned for four fifths of the circumference, and on the external surface the posterior border and a part of the internal surface had been completely destroyed for a distance of 4 cm. and replaced by grayish-white tumor growth which filled the medullary canal. On the posterior surface of the neck was an 8 mm. oval area of tumor eroding bone. On the anterior surface of the shaft, beginning 5.5 cm. above the articular surface of the condyles and extending upward 5 cm. with a width of 1.8 cm., the bone was discolored and partially resorbed, its surface roughened and eroded. On the anterior surface of the shaft immediately above the condyles were four oval areas of tumor invasion with partial bone resorption.

The right fibula had three areas of necrosis: one, globular in shape, 2 cm. in diameter, located at the head anterior to the styloid process; a second, 8 mm. in diameter, on the posterior border, 3.5 cm. above the lower extremity, and a third, 6 mm. in diameter, on the posterior surface of the external malleolus.

During cleaning, the right tibia with its corresponding fibula, was left on a table top, and the tumor growths were largely eaten out by mice, who left ragged, irregular spaces where tumor invasion had led to necrosis of the bone. The cancellous bone of both its extremities and the shaft contained twenty-one areas of tumor metastases associated with bone necrosis. These occurred most prominently as involvements of the internal tuberosity, and of the upper and lower thirds of the shaft. The largest areas were: one, 5.3 by 4.3 cm., of the internal tuberosity; a second, 6 by 2 cm., on the external surface of the shaft located 6 cm. below the articular surface of the external tuberosity, and a third, at the lower portion of the shaft, 8.5 cm. in length, involving the anterior border, the internal surface and border, and the posterior surface. The remaining shell of bone, approximately one-third the normal circumference, contained a recent transverse fracture, without separation of fragments, 6.2 cm. above the articular surface of the lower extremity. Along the external border and posterior surface of the fragments was early callus formation. On the external surface of the bone with its center crossing the line of fracture was an area of tumor growth, 2 by 1.1 cm., with bone necrosis. The other areas were distributed over all borders and surfaces of the bone and varied in size from 5 mm. to 2 cm. in diameter.

The inferior surfaces of almost the entire right internal cuneiform and the distal portion of the right scaphoid were largely resorbed, and the area was filled with a thick, putrid, purulent exudate. This bone necrosis with purulent inflammation involved an area 1.2 by 1.3 by 2.1 cm.

The articular surfaces of the long bones were nowhere involved in necrotic changes or tumor metastases. At sites of muscle and ligament attachments there were no tumor invasions of them or of bone surface from without. The periosteum was nowhere destroyed. The areas of bone resorption with their corresponding replacements by tumor metastases were in most instances surrounded by zones, a few millimeters in width, where the bone was white like dead bone, but remained as a hard, brittle rim between the area of complete bone destruction and the surrounding normal bone.

Histologic Examination.—Sections of the primary mammary tumor were typical of a scirrhous carcinoma. Material from the involved bone areas, growths in the bone medulla, of the right temporal tumor, and of the left axillary, supraclavicular and infraclavicular lymph nodes, revealed a scirrhous tumor identical with that of the breast.

SUMMARY

The bone involvements described here were more massive and widespread than any found in the available literature, and showed rarely recorded invasions of some bones, such as the malar, superior maxilla, clavicle, tibia, fibula and those of the foot.

The absence of anatomic evidence of lymphatics in the bone marrow eliminates the question of lymphatic spread to this tissue.

Osseous involvements were primarily of the bone medulla with extension to the cancellous bone and bone surfaces, making it conclusive that we here had blood borne metastases.

At all fracture sites there were attempts at callus formation (extensive at older fractures) showing slight or no periosteal involvement.

There was a frank absence of permeation of the fascial lymphatics near the invaded bones, of the liver, spleen, lungs, kidneys, suprarenals, gastro-intestinal tract, genital organs and the opposite breast.

Handley's theory of permeation for bone invasion in cancer of the breast is to be rejected in this case, for if such were absolute this patient should have been literally permeated from head to foot. We do not agree with his statement that the bones distal to the knee and elbow escape simply because the patient invariably dies before growth has spread along the deep fascia far enough to reach them.

Blood tumor emboli do exist. The more one studies the occurrence of bone involvement in cancer of the breast, the stronger becomes the conviction that the invasion of distant bones must be by blood stream. Blood metastases, which are more frequently seen in cancer of the breast than in cancer of other organs, play a more important rôle than has yet been assigned to them.

This case of an unrecognized, untreated, atrophic scirrhous carcinoma of the breast reemphasizes the important rôle of metastases and of proper complete examination of patients. The tumor was in existence sufficiently long for the occurrence of widespread secondary growths, multiple spontaneous fractures with callus formation abundant in tumor tissue, and deformities incident to bone resorption without the disease being recognized.

It is unquestionable that if proper search were made, more osseous metastases would be found. We will remain in partial ignorance of the truth on this point until there is universal improvement in necropsy service. The importance of extending necropsy examinations to include the osseous system cannot be too forcibly emphasized.

LEUKEMIA—A SARCOMA: BONE EVIDENCE

REPORT OF TWO CASES *

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The etiology of leukemia is still uncertain. To me it resembles sarcoma in a fluid medium. It acts like a malignant growth clinically. In the two cases I report, the morbid anatomy and histologic pathology indicate it to be of a sarcomatous nature. Not only do the leukemic cells invade soft parts, but the bone likewise gives evidence of destructive action of cells and fluids. Since the original suggestion of Bizzozero, many have thought it might be sarcomatous.

Osler [1] in his Principles and Practice of Medicine writes of Willcock's case, "where there were growths on the stomach and gastro-splenic omentum, leukemic infiltration of the liver, the cells being outside the capillaries." He furthermore states, "Leukemic tumors may occur in the kidneys and liver." "Bizzozero showed these tumor cells to be in active fission." "Occasionally the sternum, ribs and flat bones show great irregularity and deformity owing to the definite tumor-like expansions."

Scott [2] writes:

> The leukemias may be looked upon as nothing more or less than a fluid tumor, in which the constituent cells of the tumor are being carried throughout the system in the blood stream. Seldom is a true focus found. The more embryonic the cell, the more malignant the leukemia.

Simonds [3] writing about the Slye stock of mice says:

> Leukemia, pseudoleukemia and lymphosarcoma in these mice have distinctive features indicating that they are probably fundamentally of the same nature and probably belong among the true neoplasms.

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* From the Pathological Laboratory of the University of Maryland School of Medicine.

1. Osler, William: Leukemia, The Principles and Practice of Medicine, New York, D. Appleton & Co., 1892, p. 696.

2. Scott, R. E.: Uniform Nomenclature for Blood Counts, Mil. Surgeon **53**:321 (Oct.) 1923.

3. Simonds, J. P.: Leukemia, Pseudoleukemia and Allied Conditions in the Slye Stock of Mice, J. Cancer Research **9**:287-424 (Sept.) 1925; abstr. J. A. M. A. **86**:70 (Jan. 2) 1926.

Ewing [4] writing of leukemia says:

It has long been recognized that in certain cases the anatomic condition varies from the standard type in the direction of a true neoplasm.

Warthin's case is mentioned where "the ileum was extensively involved in a large abdominal tumor, which extended to the kidneys, stomach and regional lymphatic system." Moritz reports a similar case. Warthin [5] writing in 1907 about leukemia of fowls said:

They infiltrate, give metastases, destroy tissue, fail in inoculation and transplantation and resemble malignant tumors.

Weber [6] reports a case in a boy, aged 7, with myeloid tissue in the hilum of each kidney.

Keim [7] describes a case of "universal leukemia cutis" resembling lymphosarcoma and refers to the intimate relationship between leukemia, Hodgkin's disease, mycosis fungoides and lymphosarcoma.

Banti [8] writes:

In myeloid leukemia the vessels of bone are affected at times. The endothelium is missing and myelocytes penetrate the walls. Erosion of bone may occur. It is a systematic myeloid sarcoma of blood and lymph apparatus. Its etiology is probably infectious, though the germ has not been found.

Ribbert [9] writing of "Menschliche Zellen als Parasiten" says:

The leukocytes (white cells) of blood have a number of qualities in common with single cell parasites but do not propagate independently. Myelocytes losing their relation to the bone marrow regenerate actively in the spleen and elsewhere, acting as parasites. Leukemia—a parasitic disease—belongs to the tumors.

Fraser [10] describes the relationship of mycosis fungoides, leukemia and lymphosarcoma, saying that they are genetically related. He suggests the name "reticulum cell sarcoma."

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4. Ewing, James: Leukemia, Neoplastic Diseases, Philadelphia, W. B. Saunders Co., 1919.

5. Warthin, A. S.: Leukemia of Common Fowl, J. Infect. Dis. **4**:369, 1907; Osler's System of Modern Medicine **4**:829; Tr. A. Am. Phys., 1904; M. News **85**:1025.

6. Weber, F. P.: Leukosarcoma, Lymphosarcoma, Lymphadenoma and Infectious Nucleosis, Brit. J. Child. Dis. **18**:214-216 (Oct-Dec.) 1921.

7. Keim, H. L.: Universal Leukemia Cutis, Arch. Dermat. & Syph. **10**:579 (Nov.) 1924.

8. Banti, C. P.: Die Leukamieen, Centralbl. f. allg. Pathol. u. path. Anat. **15**:1, 1904.

9. Ribbert, Hugo: Menschliche Zellen Als Parasiten, Deutsche med. Wchnschr. **33**:329 (Feb. 28) 1907.

10. Fraser, J. F.: Mycosis Fungoides: Relation to Leukemia and Lymphosarcoma, Arch. Dermat. & Syph. **12**:773 (Dec.) 1925.

Minot and Buckman,[11] writing of Osler-Vasquez disease (polycythemia), refer to the coincident increase in the manufacture of myelocytes and red blood cells and suggest that we are dealing with a fluid tumor.

It has been seen associated with various infections—actinomycosis,[12] tuberculosis (Ellerman and Bang), fowl typhoid,[13] influenza,[14] Vincent's angina,[15] trauma.[16] The roentgen ray has produced as well as cured leukemia.[17] Flexner [18] describing lymphosarcoma holds it to be infectious in origin, although no germs were found.

Moore,[19] Schmeisser,[20] Winternitz [13] and Ellerman and Bang, experimenting with fowl leukemia, have not been able to prove its infectious origin.

There has always been some difficulty in distinguishing certain inflammatory growths from true tumors, especially those of a chronic nature, in which the toxins are less strong and longer acting. Here, the terms tuberculoma, syphiloma and granuloma are used. Leukemia, pseudoleukemia, lymphosarcoma, chloroma and mycosis fungoides have always been placed in a class together. More recent reports about such classification will be seen in studies of Keim,[7] Fraser,[10] Simonds,[8] Weber,[6] Fox and Farley [21] and Pagniez, Coste and Ravina.[17] These investigators consider them as closely allied tumors.

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11. Minot, G. R., and Buckman, T. E.: Certain Aspects of Polycythemia, Soc. Proc. Am. A. Phys., J. A. M. A. **80**:1954 (June 30) 1923.

12. Latimer, T. S.: Intestinal and Hepatic Actinomycosis Associated with Leukemia, Tr. A. Am. Phys. **11**:322, 1896. Welch, W. H.: Papers and Addresses **1**:541, 1920.

13. Winternitz, M. C., and Schmeisser, H. C.: Studies on the Relation of Fowl Typhoid to Leukemia of the Fowl, Rep. Johns Hopkins Hosp. **18**:24, 1919.

14. Blatteis, S. R.: Rep. M. Soc. State of New York, J. A. M. A. **76**:1528 (May 28) 1921.

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16. Noble, T. B.: Report of a Case of Chronic Myeloid Leukemia, Indianapolis M. J. **23**:422, 1920.

17. Pagniez, P.; Coste, F., and Ravina, A.: Myeloid Leukemia and Chloroma, Ann. de méd. **16**:381, 1924; Paris letter, Leukemia Following the Use of Radioactive Substances, J. A. M. A. **85**:1571 (Nov. 14) 1925.

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19. Moore, V.: Infectious Leukemia in Fowls, Ann. Rep. Bureau Animal Industries **12**:182; **13**:205, 1895-1896.

20. Schmeisser, H. C.: Leukemia of the Fowl—Spontaneous and Experimental, Rep. Johns Hopkins Hosp. **22**:551, 1915.

21. Fox, H., and Farley, D. L.: Relation of Aleukemic Leukemia, So-Called Pseudoleukemia and Malignant Granuloma, Am. J. M. Sc. **3**:163 (March) 1922; abstr., J. A. M. A. **78**:1419 (May 6) 1922.

Again, we are drawn closer to the consideration of leukemia with the sarcomas when we see the close relationship of its cells to the more solid myelomas. Investigators find various kinds of marrow cells constituting their reported myelomas. Symmers [22] says, multiple myelomas are neoplasms from myeloblasts, even though myelocytes and erythroblasts are implicated. Wells [23] reports a case of myeloma washed out of bone leaving enough structure behind to resemble hemangiosarcoma or perivascular endothelioma. The myeloblasts were numerous. However, one will note in his case as in some cases of leukemia, that solid tumor nodules were found elsewhere in the body (in his case, in the pleura). This case makes one think that the myeloma differs from leukemia, not so much in its cells as in its consistency. That marrow cell tumors should differ one from another in the kind of marrow cell or mixture of marrow cells comprising the tumor mass is no stranger than the same occurrence in sarcoma of soft parts. That the consistency (closer or looser organization) of the tumor cells should vary in marrow also has its counterpart in sarcomas of connective tissues.

It might be objected that marrow cells are not of the connective tissue group, but they are.

Sabin [24] writes, "Blood is not so sharply separated from connective tissue in general. In the embryo the origin of blood from connective tissues is widespread.

Maximow [25] says, "The relationship between blood cells and connective tissue is so intimate that no distinct line can be drawn between them."

Carrel grew in vitro connective tissue cells from mononuclear leukocytes. "The large mononuclears became transformed into cells which assumed the appearance of fibroblasts and developed a tendency to tissue formation."

The cells in Foot's [26] bone marrow cultures closely resembled connective tissue cells, fat cells and clasmatocytes.

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22. Symmers, Douglas: The Multiple Myelomata and Their Ability to Metastasize, Ann. Surg. **67**:687 (June) 1918.

23. Wells, H. G.: The Relation of Multiple Vascular Tumors of Bone to Myeloma, Arch. Surg. **2**:435 (May) 1921.

24. Sabin, Florence R.: Origin of Cells of the Blood, Physiol. Rev. **2**:38 (Jan.) 1922.

25. Maximow, A. A.: Relation of Blood Cells to Connective Tissue and Endothelium, Physiol. Rev. **4**:533 (Oct.) 1924.

26. Foot, N. C.: Growth of Chicken Bone Marrow in Vitro and Its Bearing on Hematogenesis in Adult Life, J. Exper. Med. **17**:43, 1913.

Margaret Lewis [27] grew blood cells in culture and macrophages, epithelioid and giant cells resulted.

There is a great deal of evidence from pathologic material that the marrow cells form adult connective tissue and also connective tissue that remains for some reason in its embryonal state and grows excessively (sarcoma). Bunting,[28] Muir and Grawitz [29] refer to such embryonal connective tissue growths in bone in cases of pernicious anemia. Von Recklinghausen and Paget in describing cases of osteitis deformans write of sarcoma developed in the bone marrow of some of their cases.

I see in ribs the cellular proliferation of the marrow resembling leukemia from long standing heart and blood vessel diseases with accompanying chronic passive congestion. In some the cells have developed further and formed fibrous tissue.

HOW EXPLAIN SARCOMA FROM MARROW, BONE, RETICULAR, ENDOTHELIAL OR OTHER MESENCHYMAL CELLS

Cells under pathologic conditions do not act just as they normally do. The nearest approach to the embryonal act of multiplying and forming tissue has been seen in cultures produced in the laboratory.

Under the stimulus of toxic materials in disease, there is seldom a perfect balance of stimulation, depression or pabulum to the cell. We see either too much or too little—not perfect physiologic satisfaction.

One can well see this in the progressive tissue changes (embryonal cell activity) in the healing of an infected wound. Cells are killed in the center of the strife against the poisons while they overgrow further away or after the bacteria or toxins have been neutralized or filtered by the surrounding tissues. We see the different effect on body cells by bacteria of the same kind but of varying degrees of virulence. The effect varies with the cell (chemical) resistance. Indeed, there are many things that tend to aid or resist the effect of toxins.

Pathologists have been striving long to discover what allows the cell, stimulated to the point of embryonal activity and appearance (in sarcoma and cancer), to remain at this stage of its life history, go no further, just continue indefinitely, multiplying as the same kind of cell or cells.

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27. Lewis, Margaret Reed: The Formation of Macrophages, Epithelioid Cells and Giant Cells from Leukocytes in Incubated Blood, Am. J. Path. **1**:91 (Jan.) 1925. Lewis, M. R., and Lewis, W. H.: The Transformation of White Blood Cells, **84**:798 (March 14) 1925.

28. Bunting: Reversion of Marrow to Embryonal Type in Pernicious Anemia, Bull. Johns Hopkins Hosp. **36**:222, 1905.

29. Grawitz, Paul: Maligne Osteomyelitis und Sarcomatose Erkrankungen des Knochen systems als Befunde bei Fallen von Pernicose Anamie, Arch. f. path. Anat. **76**:353, 1879.

Carrel [30] seems to have found the answer in the fact that the malignant cell dies quickly, leaving a toxic liquid behind, but not before causing many other cells to multiply. A huge mass of embryonal cells forming sarcoma are left behind. It is easily conceivable that this process may be simulated or actually produced by infections. I have referred to the various infections associated with the leukemias. The lymphosarcomas have likewise been said to be caused by infections.[18] Hodgkin's disease and leukemia have both been referred to tuberculosis and syphilis.

Today there is a tendency to seek for some specific infecting agent as the cause of malignant tumors. A reversion to an embryonal type of cell is commonly seen in bone as in other tissues when influenced by toxins. In several hundred necropsies, I not only see the myelocytes multiplying in infections, wood alcohol, carbon monoxide, phosphorus and cantharides poisoning but also frequently note great increase in normoblasts and bone cell multiplication. There is usually edema with enlargement and coalescence of bone lacunae.

In the chronic cases in which the irritation has been moderate over a long period of time, connective tissue formed in the marrow while the bone matrix becomes fibrillar or fibrosed. The latter condition is best seen in Hodgkin's disease, pernicious anemia, long standing tuberculosis and syphilis.

Here, in leukemia, however, we see the embryonal cell development but as in sarcoma elsewhere the cell does not complete its full development and form the kind of tissue for which it was intended.

REPORT OF CASES

CASE 1.—A white man, aged 45, was admitted, April 8, 1923, complaining of cough, bloody urine and fever (102 F.). The onset had been thirteen days before.

The patient was pale, and there were numerous petechiae over the arms, legs and body. There were fresh hemorrhages in the outer quadrant of the right eye; dulness and râles at the bases of both lungs. The abdomen was moderately distended. The spleen descended 4 cm. below the costal margin and was rounded and firm. The urine contained blood and albumin. There were a few hyaline casts. Blood cultures of the heart, spleen and liver were sterile. The red blood cells totaled 2,900,000; white blood cells, 102,000; hemoglobin, 48 per cent.; myelocytes, 88 per cent; small lymphocytes, 5 per cent; N. myelocytes, 4 per cent, and polymorphonuclears, 3 per cent.

The patient died, April 10, 1923.

At necropsy the anatomic diagnosis was acute leukemia. There were numerous petechial hemorrhages over the body, and hemorrhages over the viscera, intestine, especially along the serosa of the ileum and colon, the heart, peritoneum and kidneys. There was leukemic invasion of the spleen, stomach, intestines, lymph glands, kidney, lungs, brain and bone.

30. Carrel, Alexis: Essential Characteristics of a Malignant Cell, J. A. M. A. **84**:157 (Jan. 17) 1925.

The myelocytes had escaped from the vessels in the invaded parts and had formed large masses. They caused necrosis of the tissues involved. There was evidence of hemorrhages (red blood cells and granular pigment). In the brain and bone the nuclei of some myelocytes contained karyokinetic figures.

Fresh and decalcified sections of rib and vertebra were stained as in case 2. The cortex was thinner than normal. The bone was edematous and pigmented, with blood in scattered areas of both matrix and marrow. The lamellae were swollen and there were clefts between them, some containing myelocytes; the lacunae were enlarged; the bone cells were swollen and generally well stained. Virchow's territories were numerous. There was evidence of nuclear division in the bone cells at the margins of the cancelli and wherever these cells were close

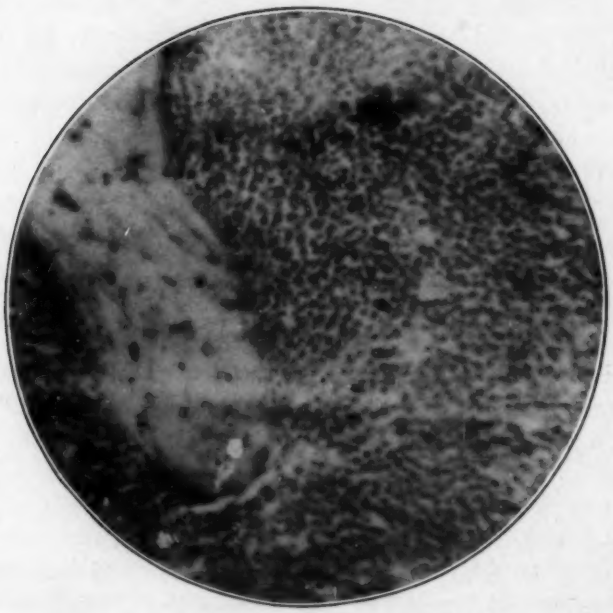

Fig. 1 (case 1).—Bone destruction by myelocytes; the bone cells are active.

to the marrow cavity and haversian canals. Many of the nuclei of the bone cells were lobulated. At times two such nuclei were seen in the same lacuna. Lacunae were frequently confluent, especially at the borders of the bone where the matrix was granular or fibrillar. Where these enlarged lacunae communicate with the marrow cavity, the bone cells were freed. Those with oval and round nuclei could be readily distinguished from the marrow cells—not so those with lobulated nuclei. There was evidence of vital and lacunar absorption of bone. Small sequestrums were seen among the marrow cells. The cancelli were grooved deeply by masses of marrow (the tumor) cells which came in direct contact with them.

The vital absorption (by fluids) was irregular in action; scattered areas appeared, the bone salts had disappeared and the matrix appeared granular.

The marrow was a mass of large cells (15 to 20 mm.) with lobulated nuclei (myelocytes) and a few with single nuclei in which nuclear division (karyokinesis) was evident.

The cells were pressed closely together, but outlined by polygonal or irregular lines of separation. Here and there was seen a prolongation of cell protoplasm, giving the appearance of a fine reticulum between the cells. There were some normoblasts and a few larger cells (30 mm.) with from four to five deeply stained nuclei. In the dense masses of cells, few red blood cells were seen and no distinct good capillaries were evident, but one could see a few rows of myelocytes separated from the main mass by a thin swollen (edematous) capillary wall. Some remnants of degenerated vessels containing granular, pigmented, poorly stained protoplasm and fragments of nuclei gave evidence of the malignant character of the surrounding cell mass. There were areas of scattered red blood cells and blood pigment. In the haversian canals, which had not been affected by the destroying cell action, vessels loaded with myelocytes were clear cut. Where the leukemic cells border bone, there was no evidence of connective tissue, reticular tissue, vessels or "endosteal" cells. The "tumor" cells tended to arrange themselves in strata, and a fine or scarce reticulum apparently connected with the bone found its way between them. They indented the bone, forming irregular rough borders and leaving a granular unstained matrix. The lacunar cells (bone cells) stained well and showed evidence of cell and nuclear division and were in direct communication with the spaces produced by the destructive action of the leukemic cells, and in many instances appeared to become an integral part of the tumor cell mass.

A careful study of this case demonstrated small hemorrhages with pigmentation in all the serous membranes, mucosa of intestine, bladder, eye-grounds, brain, bone, heart, lung, muscle and skin. The blood vessels were engorged with myelocytes. These myelocytes were of a polymorphonuclear variety as a rule, few mononuclear or polymorphonuclear leukocytes being seen.

The blood findings showed myelocytes (polymorphonuclears), 88 per cent; small lymphocytes, 5 per cent; neutrophilic myelocytes, 4 per cent, and polymorphonuclear leukocytes, 3 per cent. The granules stained bluish black by Goodpasture's stain and blue by Schultz oxidase reaction.

The bone marrow was made up almost entirely of the large polymorphonuclear myelocytes.

In the lymph glands few lymphocytes remained. The mass was polymorphonuclear myelocytes. The spleen was made up almost completely of myelocytes like those seen in the bone marrow. There were few areas of lymphoid cells. The structure was lost.

In the liver there were masses of myelocytes replacing the degenerated liver cells in large and small areas. Karyokinetic figures were seen in some of the myelocytes. Where they remained within the vessels, there was no destruction of liver cells.

In some areas the brain substance had been replaced by myelocytes infiltrating and packing the invaded areas. Karyokinesis was evident in a few cells.

In the lungs masses of myelocytes outside the vessels invaded the alveolar spaces and their walls.

The stomach, intestines and kidneys demonstrated invasion by myeloblasts in groups outside the vessels. Some of the vessel walls were degenerated and myelocytes penetrated them. There was accompanying degeneration of the invaded tissue. Bone matrix invasion and destruction with sequestration of necrotic fragments were evident. The invading masses of myelocytes were of one type, the nucleus being lobulated.

CASE 2.—A white man, aged 38, entered the hospital in December, 1920, and in October, 1921; he died Oct. 23, 1921. He complained of swelling of the stomach and loss of strength and weight, referring the present illness to a wrenching of his back in 1913.

There was a mass in the upper left quadrant of the abdomen extending to the right of the midline and into the pelvis. The inguinal, axillary and postcervical glands were enlarged. The liver dulness extended 6 cm. below the costal margin. The clinical diagnosis was chronic myeloid leukemia.

The Wassermann reaction was negative. The specimens of urine contained blood and albumin. The stool showed occult blood. The red blood cells totaled 2,860,000; the white blood cells, 576,000; hemoglobin, 55 per cent; polymorphonuclears, 45.2 per cent; polymorphonuclear basophils, 0.7 per cent; polymorphonuclear eosinophils, 1 per cent; neutrophilic myelocytes, 40.6 per cent; basophilic mononuclears, 4 per cent; eosinophilic mononuclears, 1 per cent; large lymphocytes, 4 per cent; small lymphocytes, 6.7 per cent; large mononuclears, 0.7 per cent; there were 3 megaloblasts, 8 normoblasts, and a diminished hemoglobin content. Moderate anisocytosis was noted; moderate poikilocytosis; occasional polychromatophilia, and an increase in platelets; myelocytes occurred in various stages of development.

Radium treatment was used. Jan. 8, 1921, the patient was said to be improved and was discharged. The white blood cells totaled 291,700. October 12, the patient was readmitted. He had received roentgen-ray treatments to his abdomen, but the abdominal swelling increased greatly. He suffered from dyspnea, headache, vertigo and tingling in the hands.

The spleen was much larger, extending 9 cm. to the right to the umbilicus and into the pelvis.

October 15, the red blood cells totaled 2,650,000; the white blood cells, 320,000; hemoglobin, 52 per cent; myelocytes, 76.2 per cent; lymphocytes, 2.6 per cent; large mononuclears, 0.4 per cent, and polymorphonuclears, 20.8 per cent. The ophthalmoscope revealed neuroretinitis hemorrhagica. There were strabismus of the right eye, left wrist drop and aphasia.

October 20, the patient became irrational and was unconscious for two days. October 23, he died.

Necropsy revealed chronic myeloid leukemia; myeloid hyperplasia of the bone marrow, spleen and lymph glands; myeloid invasion of the lungs, brain, intestines, stomach, suprarenals, kidney and bone.

Specimens of bone from the temporal bone and the femur were examined fresh and decalcified. Hematoxylin and eosin, carbol fuchsin, gentian violet, polychrome methylene blue and picrocarmine were used. The bones were edematous and pigmented in spots by old hemorrhages. The matrix was granular, the lamellae having lost the homogeneous linear appearance in the cancelli and the cortex nearer the medullary cavity and in the bone bordering the haversian canals. Lines of cleavage due to fluid (edema) were marked between the lamellae. Plates and rods made conspicuous by vital absorption were evident. Blood pigment in granular form was seen between the lamellae in the granular matrix and lacunae. Bone salts taking hematoxylin and fuchsin stains were not homogeneously spread but occurred in islets. Some areas did not stain at all. The borders of bone next the canals and marrow cavity were often seen in a fibrillar condition.

In the temporal bone, large areas of matrix had become fibrillar and contained divided bone cells freed from lacunar compression (the borders of lacunae having widened and become less clear). There was much evidence of lacunar absorption by massed myelocytes pressing against the bone, grooving it like osteoclasts, and sequestrated fragments of bone were seen in both the temporal bone and the femur. All the bone cells even in the denser bone, stained well. At the margins of canals and in the cancelli of the medullary cavity, they were swollen and in

Fig. 2 (case 2).—Bone erosion by myelocytes.

many instances divided. The nuclei as a rule were rich in chromatin, round, oval or lobulated in shape. Three and four nuclei were seen in some of the lacunar cells. The lacunae were enlarged and many had coalesced in the softened matrix at the bone margins and at the points of massing of the myelocytes of the marrow.

Here, too, the bone cell activity was more evident. The nuclei stained deeper. The marrow was packed with cells varying in size (8 to 20 mm.) and shape. They were seen in masses separated by polygonal lines of separation nearer the bone margins and appeared in pavement form. They were often seen separated as round, lanceolate, oval or polygonal cells. Normoblasts and Haidenhain's giant

cells were few in number. Here and there was a neutrophilic polymorphonuclear leukocyte. A few giant round cells containing two and three round, deeply stained nuclei were present. Some cells resembled Kolliker's osteoclasts. The protoplasm of the cells sometimes terminated in sharp points or staff shaped projections blunt at the end. It was coarsely or finely granular or homogeneous, staining deeply with eosin. The nuclei of the majority of the cells were round; many were oval and lobulated. Most of them were actively dividing, karyokinetic figures occurring. There were a number of capillaries congested with myelocytes incorporated in the cell mass in parts of the bone cavity; in other parts no capillaries were seen and few red blood cells were evident. It was impossible to distinguish red blood cells in the capillaries. The vessels in the haversian canals were better defined. They were crowded with myelocytes.

The temporal bone contained an unusual number of capillaries congested with myelocytes. Evidences of old hemorrhagic pigmentation were more marked here than in the femur. There was transition of good bone with smooth plates to reticulated matrix, to cellular bone, to masses of cells like those seen in sarcoma of the bone.

This case showed the invasive and destructive character of the growth as case 1 did, but the cells varied in size, shape and character of the nuclei. Many of the nuclei were round. There were few polymorphonuclear leukocytes. It was a mixed cell myelocytoma.

The splenic pulp was replaced in great part by these cells, few lymphocytes remaining. No lymphatic cells remained, nor was a lymphoid structure evident in the lymph glands. The gland was replaced by mixed myelocytes.

The cerebral substance had been broken down in areas and replaced by masses of the invading myelocytes. The pituitary gland had been almost completely destroyed. The vessels of the cerebrum, cerebellum and meninges were engorged with the mixed myelocytes (mostly mononuclear). Few red blood cells were seen. There were areas of pigmentation by old hemorrhage. There were bone destruction and metaplasia with a massing of myelocytes of polymorphous character.

In both these cases the myelocytes were seen keeping their place in blood vessels in an orderly way. But here and there—in the brain, liver, intestine, spleen, serous membranes and bone—there was a break and invasion occurred as in sarcoma elsewhere. The bone demonstrated masses of cells of a specific kind in the marrow. Vessel destruction and absence of other elements of the marrow were evident. There were no other cells (osteoblasts, endosteal or connective tissue cells) lining the bone, except in the temporal bone in which some of the spaces were lined by a vascular myxomatous tissue. Bone absorption by direct action of the tumor cells was noted, and there was reviving and proliferation of the bone cells such as one sees in other sarcomas of the bone.

The excuse for such a wide variance in the views of authors about the bone marrow cell growths is due to a lack of unanimity about the

kind of cells with which they were dealing before 1900.[31] This concerns especially the confusion of myeloma and leukemia.

In leukemia we are dealing with an actively growing cell. In fact some of the leukemias are definitely myeloblastic. Cells of like origin when in an embryonal state are always difficult to differentiate. Therefore, it appears that any of the mesenchymal cells of bone and marrow take part in the formation of the leukemia cells.

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31. Wallgren, Arvid: Ueber die Natur der Myelomzellen, Arch. f. path. Anat. **232**:381, 1921.

THE PATHOGENESIS OF BLADDER DIVERTICULA*

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ST. LOUIS

The mucosa of the urinary bladder is protected from herniation through the bladder wall when the intracystic pressure is suddenly raised by the arrangement of the three interlacing muscle layers. The outer longitudinal layer has large loosely woven bundles, as has the inner longitudinal layer; the middle circular layer has small bundles and is much more closely woven. In sectioning bladder walls, it was found that frequently there are left loose fibrous tissue pathways extending entirely through the bladder wall when the intracystic pressure is suddenly raised, bundle interruption to cause them to be oblique (fig. 1). Such pathways, I consider to be the necessary congenital etiologic factor in the formation of diverticula. The acquired factor is the intracystic pressure which varies according to the strength and irritability of the bladder wall, the strength being influenced chiefly by any degree of muscular hypertrophy secondary to an obstruction. Opinion in late years distinctly favors a combination of congenital and acquired factors in the pathogenesis of these malformations. Day and Martin,[1] Hinman,[2] Judd,[3] Lower,[4] Judd and Scholl,[5] Thomas [6] and Young [7] favor such a combination of factors. Watson [8] studied the vesical cavity in its fetal development and found factors which may be of importance in determining the formation of diverticula, namely, "invaginations bridging the vesical cavity, particularly near the lateral margins of the trigone."

The point that bears the greatest strain is near the border of the trigone. It is here also that many of the muscle bundles are larger, less numerous, and find their attachment, so lessening the elasticity of this area. There are more fibrous tissue pathways in this region. The

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1. Day, R. V., and Martin, H. W.: Diverticula of Urinary Bladder, J. A. M. A. **84**:268-272 (Jan. 24) 1925.

2. Hinman, F.: J. Urol. **111**:207-242 (Aug). 1919.

3. Judd, E. S.: Tr. Am. Surg. A., 1918, pp. 519-554.

4. Lower, W. E.: Diverticula of Urinary Bladder, Arch. Surg. **3**:38-55 (July) 1921.

5. Judd, E. S., and Scholl, A. J.: Surg. Gynec. Obst. **28**:14-26 (Jan.) 1924.

6. Thomas, G. J.: Surg. Gynec. Obst. **23**:278-384 (Oct.) 1916.

7. Young, H. H.: Johns Hopkins Hosp. Rep. **13**:404, 1906.

8. Watson, E. M.: Developmental Basis for Certain Vesical Diverticula, J. A. M. A. **75**:1473-1474 (Nov. 27) 1920.

combination of the two factors, lessened elasticity and more potential openings, favors the formation of diverticula, where clinically they are most often found. The female bladder differs markedly in its attachment at the trigonal border. Due to the absence of prostatic and seminal vesicle fixation, there is less loss of elasticity and consequently diverticula are less often found in the female than in the male, even with the same apparent degree of obstruction or bladder wall decompensation, as measured by the quantity of residual urine. That they do occasionally occur is due to an unusually large and lax fibrous tissue pathway which will permit a low intracystic pressure to cause the herniation.

The lessened elasticity and fixation at the trigonal border can readily be shown in a normal youth's bladder by overfilling it to the point of

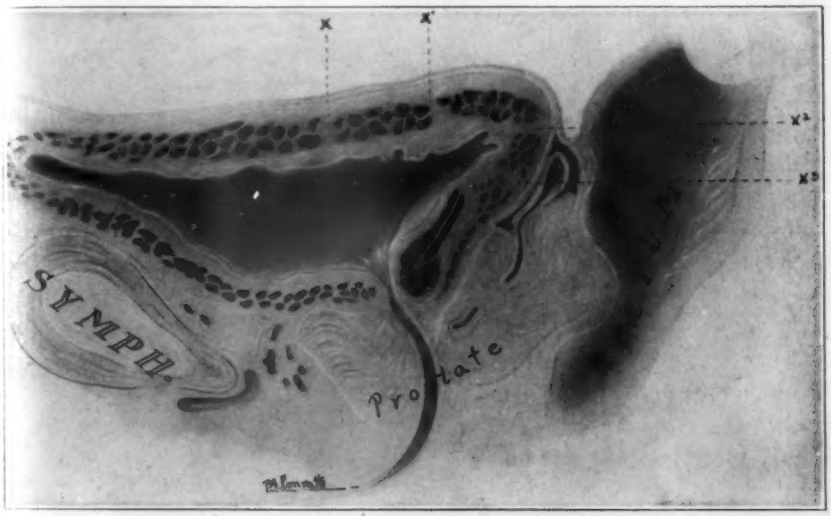

Fig. 1.—X-X^1-X^2-X^3, loose fibrous tissue pathways through the bladder wall, semidiagrammatic representation.

spasm; sharp outstanding muscle bundles bounding open mouthed cellules will occur in this region, some surprisingly deep when the spasm is at its greatest. This condition lessens markedly toward the dome and lateral walls, as the muscle bundles there are more finely divided. When an obstruction occurs, the back pressure increases, which does not mean that the intracystic pressure is constantly raised, but that a greater intracystic pressure is necessary at the time of emptying the bladder. It is raised for longer periods in the early days of its residual urine or infection. The effect of a gradually raised pressure with the congenital factor present is shown cystoscopically in figure 2 by three successive stages in the herniation of the mucosa of a youth's bladder and the

final formation of the mouth of a ballooning cellule.[9] In 1 the pressure was low, in 2 slightly raised, showing a dimple, and in 3 the mucosa and submucosa are herniated through a fibrous tissue pathway, showing a typical diverticulum mouth. At this last stage, 3, a cystogram was

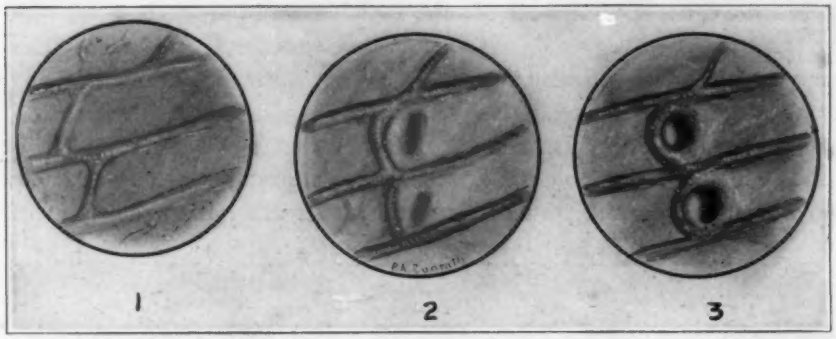

Fig. 2.—Cystoscopic views of the successive stages in the herniation of a ballooning cellule in a normal youth's bladder, brought about by gradually increasing the intracystic pressure.

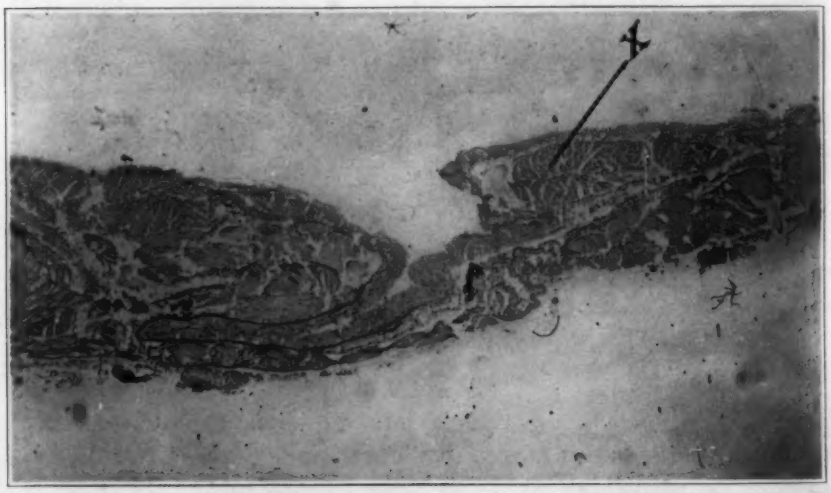

Fig. 3.—Cross section with the bladder at rest of a ballooning cellule or early diverticulum, such as seen in figure 4.

made (fig. 4) which was diagnosed early diverticulum by the radiologist. Cross section of such a ballooning cellule or early diverticulum, though somewhat more advanced, is shown in figure 3, in which the sac of the cellule, with the bladder in relaxation, lies between the middle and outer

9. Rose, D. K. South. M. J. **19**:206-212 (March) 1926.

muscle layers. In this figure, it is easy to visualize how with increased pressure the cellule would assume exactly the form that it does in the cystogram (fig. 4).

Intracystic pressure has been measured by the use of the cystometer,[10] an instrument which records the pressure and the bladder fluid content simultaneously. Types of pressure influencing bladder herniae are shown in figures 5, 6 and 9, in which the abscissas represent cubic centimeters of fluid and the ordinates the intracystic pressure in millimeters of mercury. The asterisks mark the points at which, in the

Fig. 4.—Ballooning cellule seen cystoscopically in stage 3 of figure 2: The "neck" of the malformation shows the thickness of the bladder wall.

process of gradually filling the bladder, the patient first feels a desire to void, and *v* in figure 5 the point of involuntary emptying around the catheter or cystoscope. The lower curves (marked with dots) are formed by the contraction of the bladder wall alone; the upper curves (marked with squares) are formed by all possible forces combined, i. e., contraction of bladder wall, pressure of abdominal wall, and by the descent of the diaphragm. In figure 5 (a normal curve) the intracystic pressure rises gradually to 20 mm. of mercury when the bladder con-

10. Rose, D. K.: Tr. Urol. Sect., A. M. A., 1926; J. A. M. A. **88**:151 (Jan. 15) 1926.

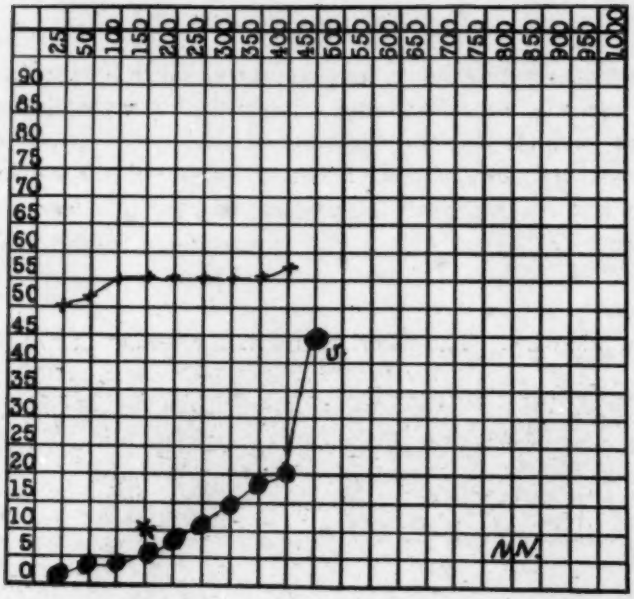

Fig. 5.—Cystometric chart of a normal bladder pressure, showing the pressure used when cystogram (fig. 7) was made.

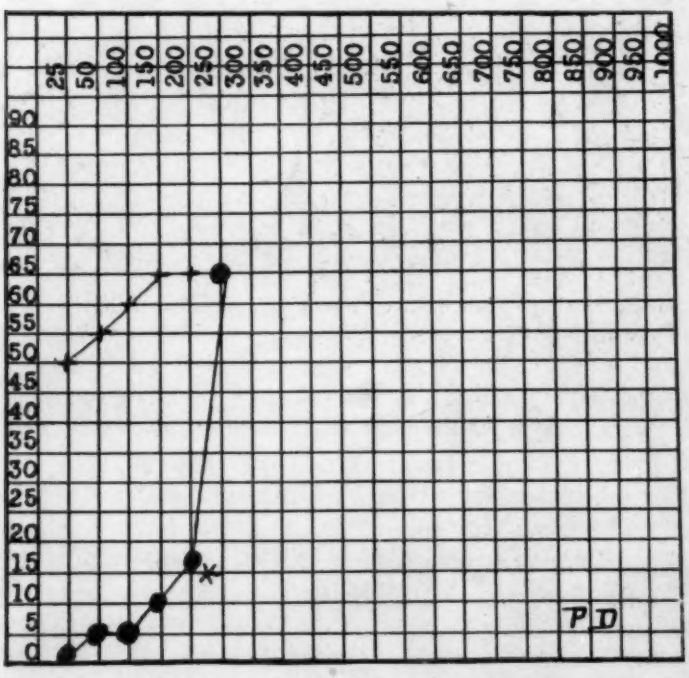

Fig. 6.—Cystometric chart in a bladder with early obstruction and hypertrophied wall, showing intracystic pressure at which cystogram (fig. 8) was made.

tracts and empties itself at 45 mm. of mercury with a content of 450 cc. of fluid. In figure 6 (representing early bladder wall hypertrophy caused by beginning prostate enlargement, with no urinary infection), as soon as the desire to void is felt, marked with an asterisk on the cystometer chart, the hypertrophied bladder wall causes spasm, raising the pressure abruptly to 65 mm. of mercury with a content of 250 cc. of solution.

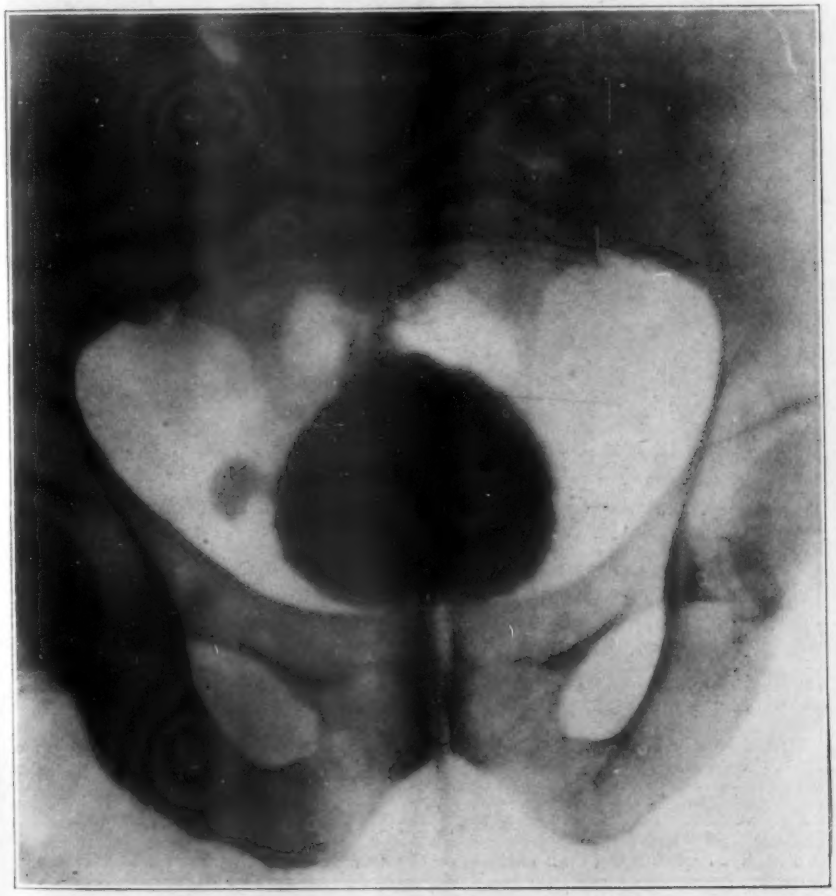

Fig. 7.—Early diverticulum.

These two charts can be visualized in cystogram by making the roentgen-ray exposure the moment the intracystic pressure and bladder content (15 per cent solution of sodium bromide) are at their limits, i. e., in figure 5 the bladder empties, and in figure 6 there is intense pain due to spasm of the bladder wall. Figure 5 is represented by cystogram (fig. 7) and figure 6 by cystogram (fig. 8). In the former, by the

gradual filling, the ballooning cellule is moderately distended and shows its pointed dependent portion, which is due to the muscle bundles of the outer layer still covering and compressing this malformation against the bladder wall. In figure 8, the bladder content is 200 cc. less; the pressure, however, is markedly raised and the ballooning cellule correspondingly dilated.

Fig. 8.—Cystogram of an early diverticulum with the bladder wall in spasm.

The size of a diverticulum indicates the length of time a malformation has been completely herniated through the bladder wall and subjected to a raised intracystic pressure *before infection supervened;* the size of its mouth up to a certain point follows the same rule. The orifice after having pushed back muscle bundles in its dilatation becomes so fortified that it cannot become larger, but the sacs cease enlarging only when their walls become fixed and lose their elasticity by the laying down of fibrous tissue.

In neurogenic bladders, the cystometric determinations demonstrate the reason for the infrequent occurrence of diverticula found, even when there is a large residual urine present due to the neurogenic obstruction. The marked difference between figures 6 and 9, the latter a neurogenic bladder wall curve in a case of tabes dorsalis, shows that the pressure is not due to the amount of fluid but to the strength and contractility of the wall, as we would expect. In figure 9, after the first desire to void is noted (marked by an asterisk as 600 cc.), the bladder wall can only raise the pressure 15 mm. of mercury. This type of pressure is not sufficient

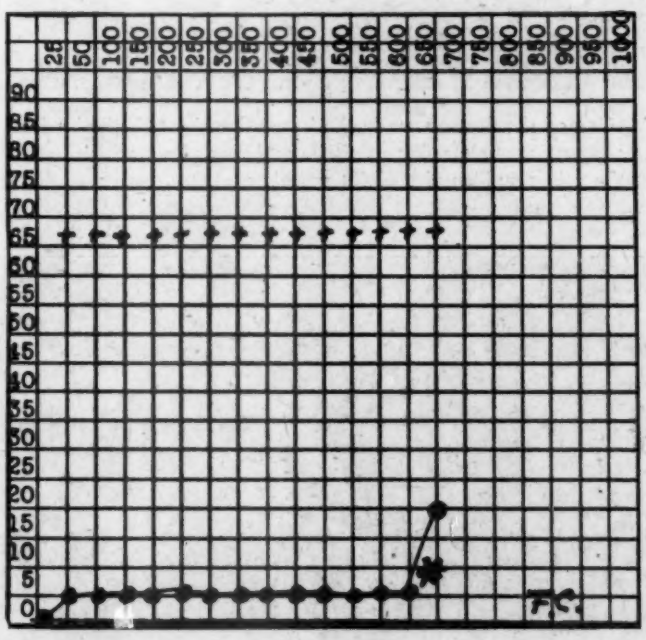

Fig. 9.—Cystometric chart showing the intracystic pressure in a neurogenic bladder (tabes dorsalis).

to cause the mucosa to herniate, even if a fibrous tissue pathway located near the trigon were present.

Herniation of the mucous membrane through the wall is shown in successive stages by figures 10 and 3; in the former a small permanent fold points toward the pathway through which a diverticulum would form if it were not interfered with by a strong outer muscle coat. The next stage (fig. 3) shows the continuation of this fold in its dissection through the bladder wall. It is still withheld from breaking through by the outer muscle layer, but the sac now lies between the middle and outer muscle layers. In the absence of infection it remains elastic and so by dilatation forms a ballooning cellule or small diverticulum when-

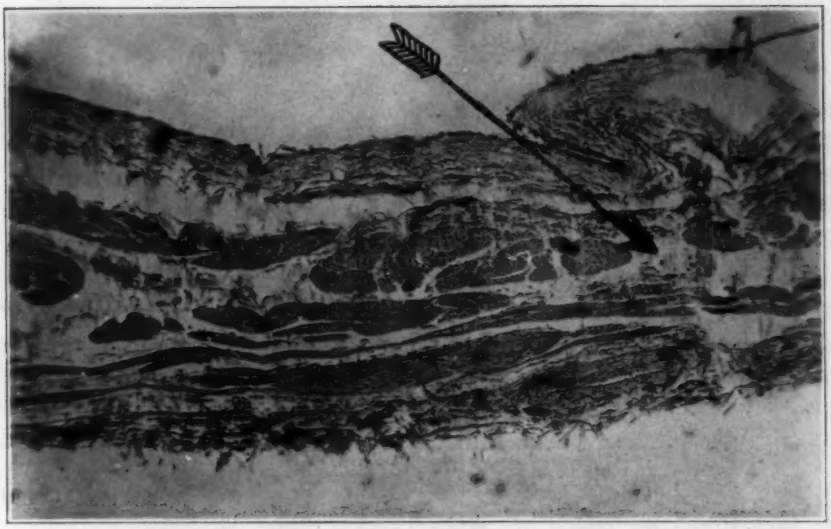

Fig. 10.—One of the first stages in the herniation through the bladder wall; the arrow points to an incomplete fibrous tissue pathway along which herniation takes place when there is a sudden rise of intracystic pressure.

Fig. 11.—Different stages of herniation in a bladder with long standing obstruction and infection.

ever the intracystic pressure is sufficient. Infection will later gradually fix the malformation so that its wall will become permanently and either partially or entirely without the bladder wall surface, at which time we have the true small diverticulum formation, and at which time only does it become a surgical condition. Before such fixation, removal of the obstruction alone is indicated.

Fig. 12.—Fully formed diverticulum with muscle layers drawn to a point at *X*, the remaining diverticulum sac being of fibrous tissue with an occasional adherent muscle bundle.

To explain the formation of diverticula in children, it is only necessary to consider that the larger the congenital defect in the bladder wall the less intracystic pressure is required and the shorter the duration necessary for this normal intracystic pressure to continue in order that a diverticulum may be formed.

The effects of an increased intracystic pressure of long duration in a bladder with definite predisposition to herniation is shown in figure 11. There are lateral dissections under both muscle bundles and mucous membrane (in upper half); a well formed diverticulum (in middle), with its puckered orifice due to the contraction of its mouth, which shows definitely as thickened muscle bundles, and a small ballooning cellule (near the lower margin) with a round well formed mouth but with floor still incompletely dissected through the bladder wall. This type of specimen is obtained by filling a bladder removed at necropsy with a 10 per cent solution of formaldehyde to the point, both as to intracystic pressure and fluid content, that theoretically was present when it contracted to empty itself in life.

The last stage in the herniation, or the completed diverticulum, is shown in cross section in figure 12, in which all three muscle layers are drawn to a point (X), the fibrous tissue sac extending on, fastening a few stray muscle bundles to itself by its inflammatory process. This idea agrees with that of Hinman,[2] who in his analysis of 205 cases states that the classification of Englisch [11] and Rathbun [12] of "true and false diverticula," the former showing all coats of the bladder wall and the latter only the mucous membrane, cannot be made. The thickness of the diverticulum wall is undoubtedly due to the inflammatory processes, except in true congenital malformations, such as urachal remnants and ureteral buds.

SUMMARY

1. Bladder diverticula are congenital to the extent that a loose fibrous tissue pathway must be present through the bladder wall before the acquired factor, namely, a raised intracystic pressure, can herniate the mucosa.

2. The occurrence of diverticula as to age and sex is determined by the size of the unprotected areas through the bladder wall, the intracystic pressure, and the variations in the fixation of the trigonal area in male and female. In females and children the normal intracystic pressure will only herniate the mucosa through a bladder wall defect of unusually wide diameter.

3. Diverticula, so diagnosed by cystogram and cystoscopic examination, in apparently normal youths' bladders, have been produced by simply increasing the intracystic pressure sufficiently to force the mucosa through an unsuspected loose fibrous tissue area in the wall.

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11. Englisch, J.: Arch. f. klin. Chir. **73**, 1904.
12. Rathbun, N. P.: Surg. Gynec. Obst. **29**:28-32 (July) 1919.

4. Cystograms of diverticula should be made with known quantities of fluid and known intracystic pressure so that the elasticity of the sac can be determined, as potential diverticula (ballooning cellules) indicate only a removal of the bladder obstruction.

5. This theory of herniation can account for all types of bladder diverticula except ureteral buds and urachal remnants.

SYPHILITIC ULCERATIONS OF THE STOMACH *

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A perusal of the American literature of the last ten years on the pathology of the stomach would indicate that gastric syphilis was a comparatively common condition. Many clinicians report a series of from twenty to thirty cases of syphilis of the stomach and certainly give the impression that not only is gastric syphilis common, but that it is also readily recognized clinically as well as at operation. A few critical reports have inadequately dispelled this idea, and up to the publication of the splendid review of Hartwell [1] there has been no critical analysis of the subject or of the reported data on syphilis of the stomach.

Gastric syphilis is not frequent and its clinical and histologic recognition is by no means simple or easy. Among the great number of gastric lesions seen by us within the last few years, there were three cases of peculiar ulcerations which by their gross anatomic features could be differentiated from the simple peptic ulcer on one hand and from ulcerated neoplasm on the other, without difficulty. These cases suggested to us the possibility of a specific lesion, particularly syphilis. Certain clinical features seemed to support this conception. However, a detailed histologic study compelled us to reconsider our preliminary diagnosis. We report these cases with the question in mind as to whether it is possible to determine the exact nature of such lesions from their anatomic and histologic appearance, without the demonstration of the etiologic organisms.

REPORT OF CASES

CASE 1.—*History.*—W. E. P., a married, white man, aged 46, was admitted to the service of one of us (C. G. H.) complaining of abdominal distress and belching of gas for nine months previous to admission. Seven years before he had had trouble with his spine, his extremities being paralyzed for six months; this was diagnosed as tuberculosis of the spine. In the hospital the patient said that he now had no venereal infection but said that he had had a chancre fifteen years before and had been treated for this condition. Nine months before admission he filled up with gas immediately after taking food. Later the gas came on about one hour after meals. He had a sense of weight after eating. There was no

* From the Department of Laboratories and the Department of Surgery, New York Post-Graduate Medical School and Hospital.

1. Hartwell, J. A.: Ann. Surg. **81**:767 (April) 1925.

actual pain. He also had frequent heartburn; induced emesis gave relief. He had little nausea, but he was constipated. Six weeks before admission he had had an attack of dysentery caused by a water-borne epidemic.

Operation (report of C. G. H.)—An infiltrating medullary carcinoma involved the terminal portion of the stomach and pylorus. The pyloric passage admitted

Fig. 1 (case 1).—Diffuse and nodular infiltration of the submucosa in syphilis of the stomach; × 30.

a quill only. The stomach was slightly dilated and contained coarse food fibers, peas, bismuth and broken-down vegetable material. The glands along the greater curvature were enlarged but seemed hyperplastic rather than neoplastic. There were no enlarged glands along the lesser curvature except just at the pyloric juncture. The pancreas was normal; the mesocolon was normal. The liver showed many scars with considerable fibrosis, suggesting a syphiltic liver rather than

one of the hypertrophic cirrhotic type. The patient died the third day. Permission to perform a necropsy could not be obtained.

Pathologic Examination.—The specimen was of the pyloric end of the stomach, 40 mm. along the lesser curvature and 100 mm. along the greater curvature. There was a portion of omentum attached to the greater curvature. The

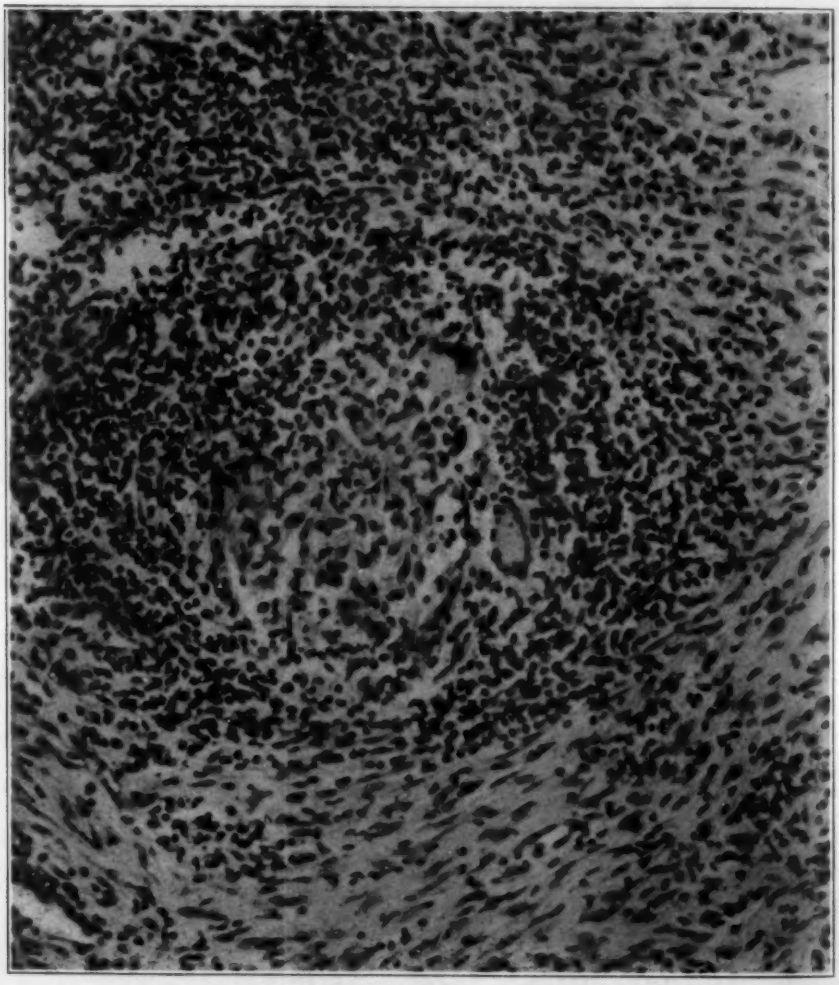

Fig. 2 (case 1).—Miliary gumma surrounded by plasma cells; ×230.

lumen was 20 mm. in diameter at the smaller end. On section there was an irregularly outlined ulcer, approximately 30 by 13 m. in area, extending upward along the greater curvature from the pylorus. This ulcer was rather shallow; its base was fairly firm. The muscle and fibrous tissue beneath the ulcer was 13 mm. thick. In the omentum there were small firm spots, apparently lymph nodes. The largest lymph node was about 18 mm. in diameter. It appeared normal.

Microscopic Examination.—Sections from the wall of the ulcer showed the surface epithelium of the mucosa desquamated. The pyloric glands were here and there dilated and branching. The stroma of the mucosa showed extensive infiltration with polymorphonuclear leukocytes and plasma cells. At the base of the mucosa were several ill defined nodules composed of large spindle-shaped

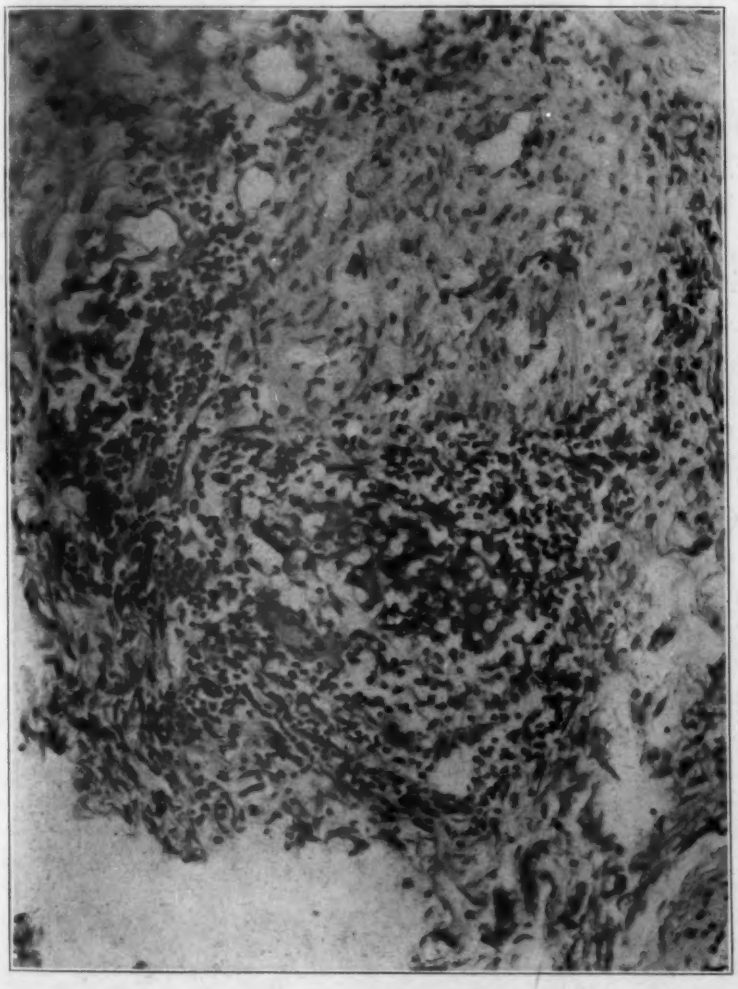

Fig. 3 (case 1).—Fibrous nodule and miliary gumma; hematoxylin-eosin stain; × 230.

cells with pale nuclei intermingled with lymphocytes, a few polymorphonuclear leukocytes and plasma cells. Within these nodules there was a small number of giant cells with peripherally arranged dark nuclei. The submucosa and the underlying muscularis showed edema, and the muscle fibers of the muscularis were separated by strands of polymorphonuclear leukocytes, lymphocytes and plasma cells.

Within the region of the ulceration the mucosa and muscularis mucosae appeared completely destroyed. The gastric wall consisted here of a superficial layer of very cellular granulation tissue beneath a layer of edematous connective tissue, and a muscular coat on which two layers perpendicular to each other could be distinguished. The superficial layer of the submucosa appeared as a granulation

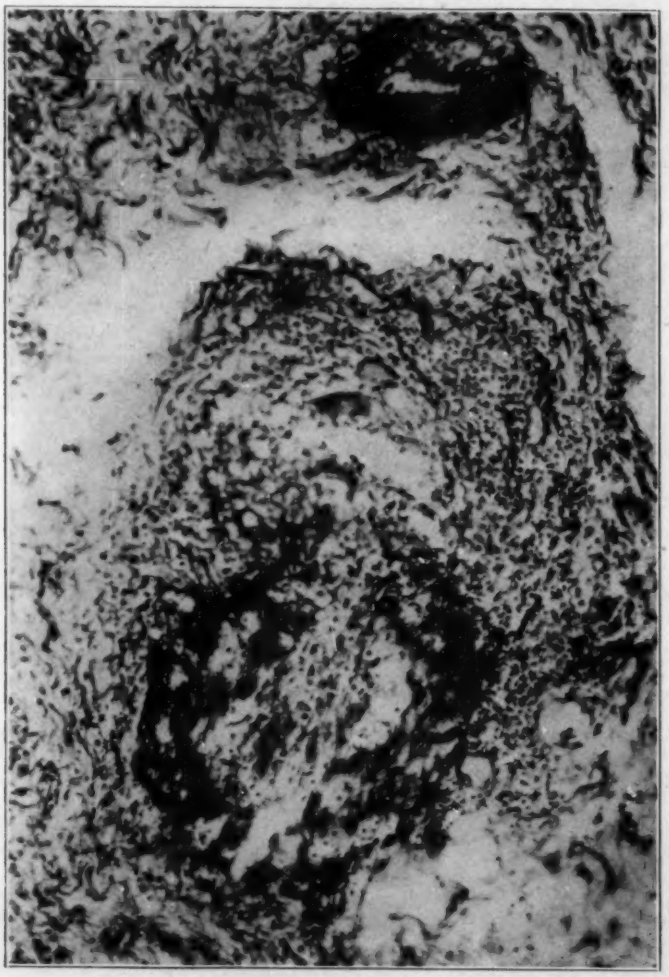

Fig. 4 (case 1).—Fibrous nodule and miliary gumma; Weigert's elastica stain; × 230.

tissue heavily infiltrated by lymphocytes, numerous plasma cells, polymorphonuclear leukocytes, fibroblasts varying in number in different places and occasionally by large mononuclear cells with pale nuclei. The granulation tissue was rich in newly formed capillaries and small arteries. Within the layer beneath, which was composed of a loose arrangement of connective tissue fibrils, there were marked edema and an interfibrillar infiltration, with plasma cells in predominant numbers, occasional polymorphonuclear leukocytes and lymphocytes. Even within

the low power magnification a perivascular arrangement of the plasma cells could be readily recognized. Because of this arrangement, the infiltration appeared to be of a nodular type. The center of such nodules occasionally showed a proliferation of large spindle-shaped cells with pale nuclei. In one of these nodules giant cells of the Langhans type were detected.

The cellular infiltration extended in a more diffuse manner into the muscular coat, but even here the nodular arrangement was not entirely missing and generally showed the same perivascular grouping around small blood vessels. Within the nodules of the muscular coat, however, lymphocytes were predominant, whereas the diffuse infiltration was made up chiefly of plasma cells.

The conspicuous perivascular type of the cellular infiltration necessitated a further detailed study of the medium sized and small blood vessels. The cells composing the granulation tissue were not restricted to the perivascular areas. They invaded the blood vessel walls, starting from the adventitia and advancing to the lumen with simultaneous destruction of elastic coats. In such blood vessels the intima was markedly thickened and the lumen narrowed. Numerous veins were completely obliterated, their lumina filled by granulation tissue, with Langhans' giant cells. In the sections stained with hematoxylin-eosin such vessels resembled tubercles. Their true nature was revealed by application of Weigert's elastic stain.

Sections from lymph nodes showed enlarged lymph follicles with large germinal centers.

Impregnation with Levaditi's silver method did not reveal spirochetes. A stain for tubercle bacilli remained negative.

The diagnosis was syphilis of the stomach.

CASE 2.—*History.*—D. G., an unmarried colored man, aged 25, was admitted to the medical wards, Dec. 16, 1924, complaining of abdominal distention and discomfort after meals for nine months previous to admission. He had vomited after meals for one month. In the past there had been slight occasional tachycardia and palpitation. Gonorrhea eleven months previous to admission was followed by buboes. He said that he had not had syphilis. He had suffered from gas in the gastro-intestinal tract and heartburn for the last nine months, and he was constipated. His appetite was good. Nine months before admission, the patient began to suffer considerable abdominal distress, gas, heartburn, distention and belching of gas about fifteen minutes after eating. At first these symptoms following meals occurred only occasionally, but they had recently become progressively more frequent and intense. The belching of gas relieved this distress; his appetite was good, but he limited his diet because of fear of distress. One month before admission he began vomiting at the height of distress. The vomitus consisted of recently ingested food and at first relieved the distress. Two weeks before admission to the hospital it appeared rather black, but no bright red blood could be noticed. The bowels were constipated. Two weeks before admission, he had tarry black stools, lasting for several days. He had lost 20 pounds (9 Kg.) in weight during the last nine months with a simultaneous loss of strength.

Laboratory Examinations.—The red blood cells totaled 4,162,000; the white blood cells, 5,050; the hemoglobin content (Sahli), 73 per cent; polymorphonuclear leukocytes, 62 per cent. There was a moderate trace of albumin in the urine; the sediment showed numerous pus cells. An Ewald meal showed free hydrochloric acid 23.6, combined 15.6, total 48.6. Blood in the gastric contents was strongly positive. The blood chemistry was normal. Four Wassermann reactions, one provocative, were negative. Tested for blood, the feces were strongly positive.

Roentgen-Ray Examination (Dr. W. H. Meyer).—A rather wide gap of poor, irregular filing was seen in the immediate prepyloric region. This defect measured about 1 inch (2.5 cm.) in length; the canalization was narrow and irregular. The bulbus of the first portion of the duodenum was large and irregular. Peristalsis was sluggish, these peristaltic waves disappearing in the antrum at the point of

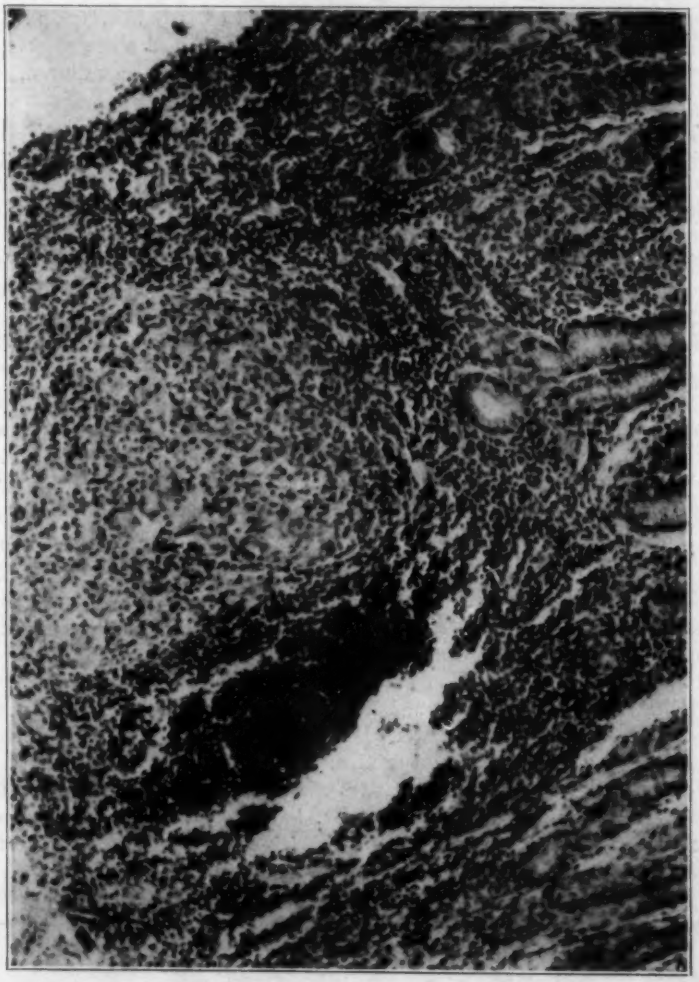

Fig. 5 (case 2).—Epithelioid cell tubercle originating within a lymph follicle of the mucosa in tuberculosis of stomach; × 200.

the defective filling mentioned above. Evacuation was delayed with a medium sized gastric and duodenal residue at the end of six hours. Otherwise, motility was comparatively normal.

The pathologic conditions were those of partial pyloric obstruction. The defective filing was rather suggestive of infiltration; whether primary pyloric or secondary to retroperitoneal or pancreatic involvement was not determinable. The lesion did not have the characteristics of a primary pyloric malignancy, but simu-

lated the condition usually observed in syphilis. Comparative examination after administration of atropine confirmed the opinion that there was benign infiltration.

In spite of four negative Wassermann reactions and a negative history, the diagnosis of syphilis of the stomach was made.

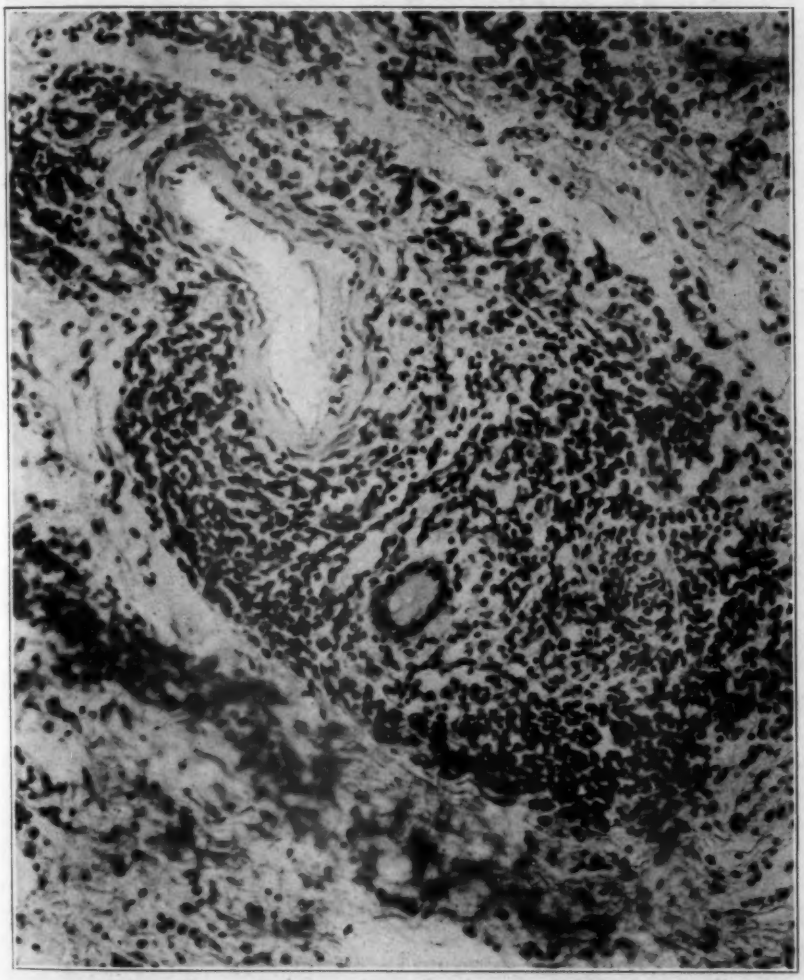

Fig. 6 (case 2).—Miliary tubercle within a perivascular plasma cell infiltrate; × 230.

Course.—The patient's condition became gradually worse; he vomited often, and the symptoms of pyloric obstruction made it necessary to perform an exploratory operation. This was done by Dr. John F. Erdmann, Jan. 2, 1925. An ulcerated, infiltrated pyloric end of the stomach was removed with subsequent Polya anastomosis. The postoperative diagnosis was gastric ulcer with infiltration and stenosis.

On January 5, examination of the chest revealed a double bronchopneumonia. The temperature was 103.6; pulse, 160, and respiration, 60. The patient was

restless and weak; the pneumonia grew progressively worse; the patient became more and more exhausted, and died, Jan. 8, 1925. No necropsy was obtained.

Pathologic Examination.—The specimen was a piece of the prepyloric portion of the stomach, which had been opened. It measured 140 by 120 mm. in diameter. There was adherent omentum, in which numerous glands the size of a pea were found. Near the pyloric end was an ulceration of irregular outline, which con-

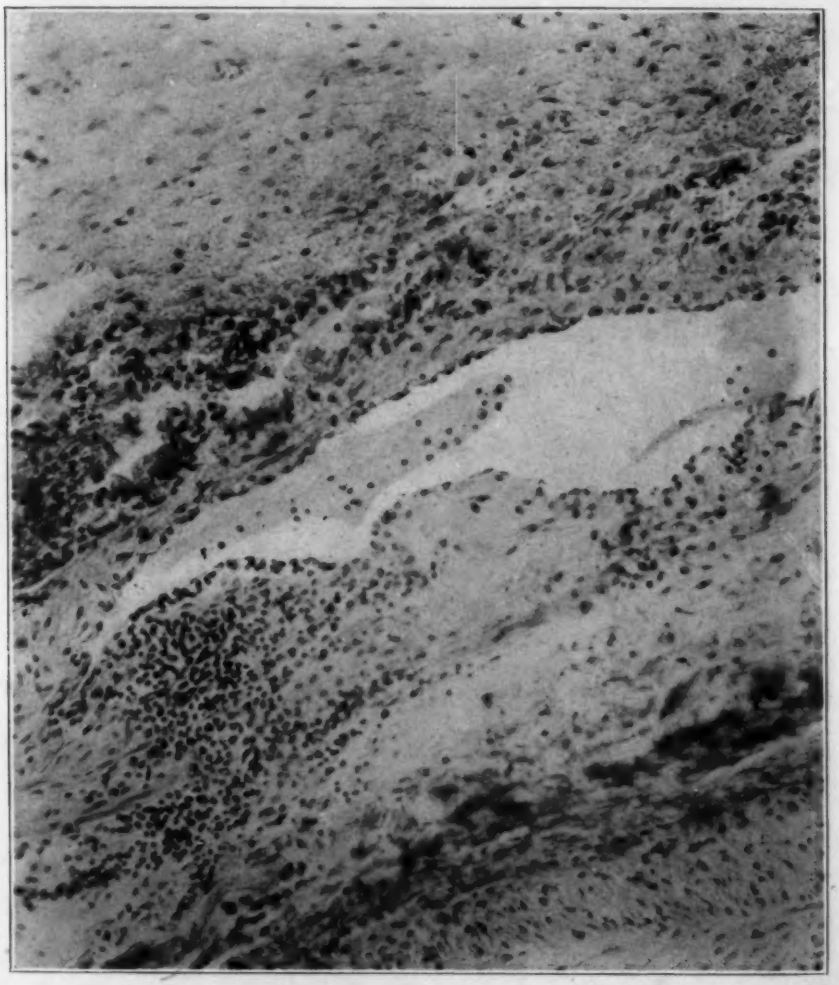

Fig. 7 (case 2).—Focal phlebitis; × 180.

sisted of two parts connected by a narrow furrow, one part measuring 50 by 20 mm. and the other 30 by 20 mm. The margin was in part smooth, partly, however, serpiginous, and it was slightly overlapping. The base of the ulceration, which was about 4 mm. deep, was grayish with small areas of granulation tissue. The submucous coat was considerably thickened and edematous. The remainder of the mucosa was smooth and showed hemorrhagic spots. On the serosa corresponding to the areas of the ulcer there was a hemorrhagic area the size of a

silver dollar with a central flat nodule which appeared to be connective tissue. The glands were grayish and edematous.

Microscopic Examination.—Sections from the wall of the ulcer showed desquamation of the surface epithelium. In parts the pyloric glands were distintegrating. The interglandular areas were infiltrated with a granulation tissue composed of numerous plasma cells; many epithelioid cells; polymorphonuclear

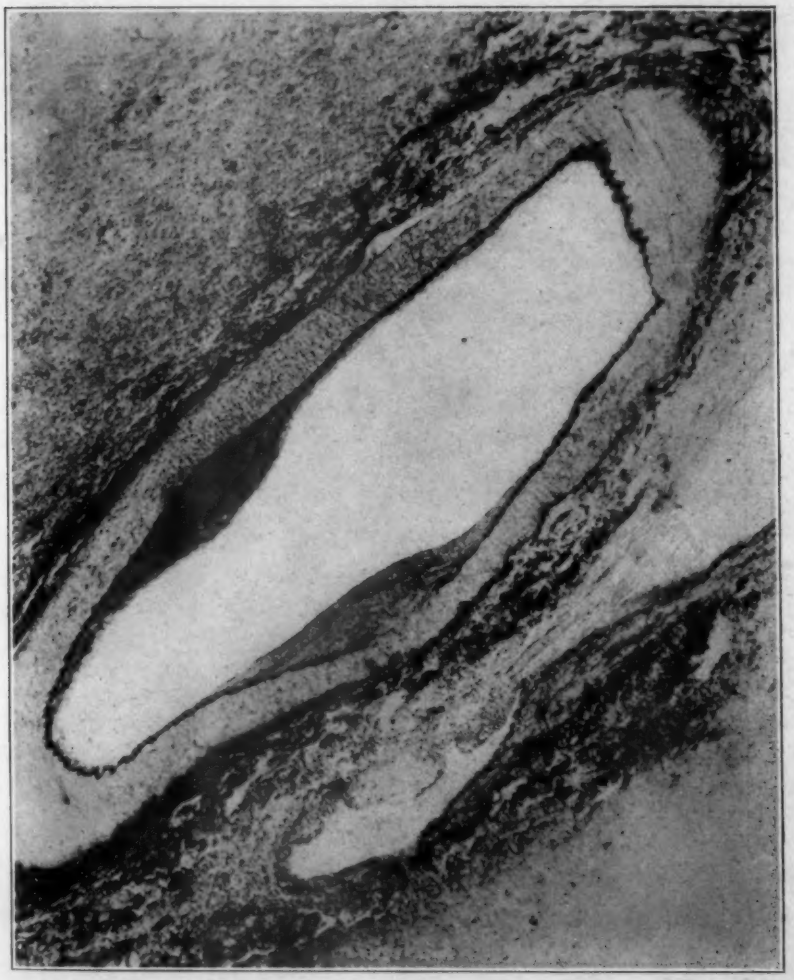

Fig. 8 (case 2).—Focal destruction of the lamina elastica with endarteritis; ×30.

leukocytes; small round cells; eosinophils; a number of cells aggregated in nodular form, composed of lymphocytes, and epithelioid cells, with occasional giant cells in the somewhat rarefied center. Close to the ulcer the mucosa suddenly ceased and in some areas the descent was steep. Other sections, however, showed an undermined overlapping mucosal border, the defect being caused by the absence of muscularis mucosae, which in its intact portions from the wall of the ulcer appeared edematous and infiltrated by similar granulation tissue.

The submucosa, the muscularis mucosae and the serosa were infiltrated by diffuse granulation tissue and tubercles.

The base of the ulcer was formed by the submucous; the mucosa and the muscularis mucosal were absent. The submucosa was covered by a layer of necrotic tissue. The width of the submucosa was much greater than in the vicinity of the ulcer; this was because of the extensive cellular infiltration, which was partly

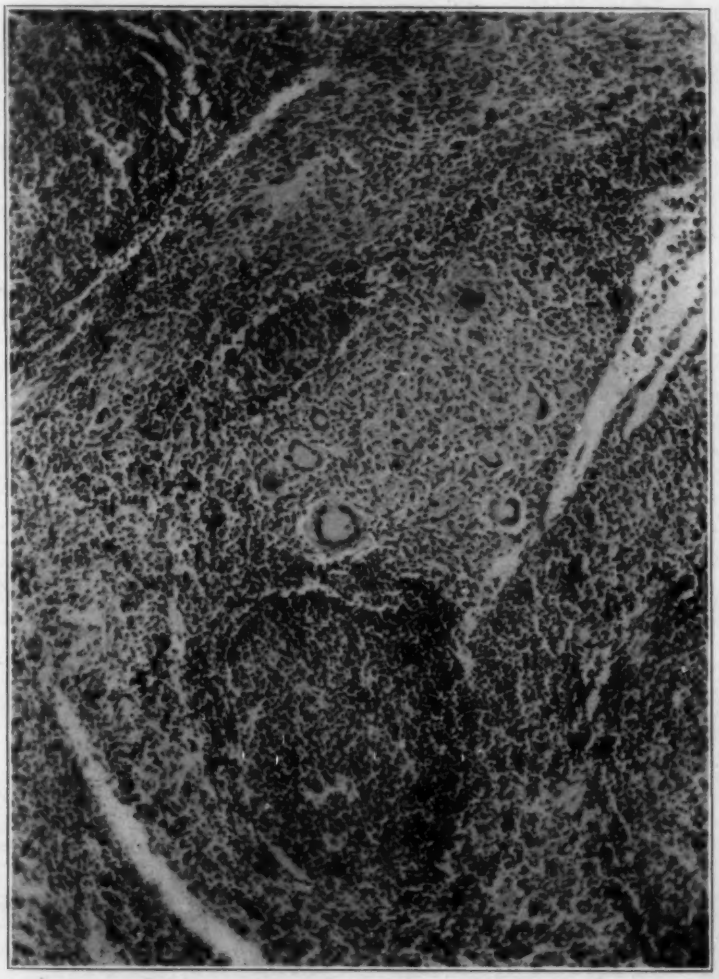

Fig. 9 (case 2).—Tubercle within a regional lymph node.

diffuse and partly in the form of tubercles and edema. Here conspicuous perivascular arrangement of the cellular elements was noticed, plasma cells, lymphocytes and epithelioid cells being the main constituents. Occasionally, within the perivascular infiltrate composed of plasma cells were small nodules composed entirely of epithelioid cells, with an occasional giant cell of the Langhans type.

The muscularis showed marked edema; diffuse cellular infiltration and tubercles were prevalent, with occasional Langhans' giant cells. The serous coat also showed tubercles and lymphocytic infiltration.

The blood vessels throughout showed severe changes. All coats of the numerous arteries as well as the venous channels were pervaded by granulation tissue and some of the blood vessels showed destruction of their coats. In such vessels the intima showed thickening, the lumen was irregularly narrowed and the continuity of the elastic coats was interrupted. However, no complete obliteration of the blood vessel lumen was noticed in any of the numerous sections.

Concerning the lymph nodes, the periglandular fat and connective tissue showed several tubercles and also diffuse lymphocytic infiltration. The lymph glands proper showed large lymph follicles with large germinal centers and several tubercles with Langhans' giant cells and caseation.

Guinea-pigs were inoculated and direct cultures and smears made. The antiformin method was applied to numerous frozen sections. Levaditi impregnations for spirochetes were done. All were negative.

The diagnosis was ulcerative tuberculosis of the stomach.

CASE 3.—*History.*—P. W., a white woman, aged 37, who had been married ten years, was admitted, Feb. 27, 1925, to the service of one of us (C. G. H.), complaining of pain in the right upper quadrant of one year's duration. She had no children and had had no pregnancies. For the last year the patient had had frequent attacks of pain in the right upper quadrant, the last attack occurring on the day previous to admission. The pain was sharp at times, dull at others, radiating to the back but not to the shoulders; it was accompanied by vomiting and relieved after vomiting. The patient vomited three times on the day of admission. The vomitus consisted of yellowish fluid. The patient had been troubled by gas and indigestion which were aggravated by certain foods, such as cabbage. The bowels were constipated; the stools were not clay colored. There was no jaundice. She had lost 25 pounds (11.3 Kg.) in weight in one year. The family physician stated that the patient's husband had a positive Wassermann reaction and was under treatment at this time. On physical examination a tumor was palpable in the epigastrium a little distance to the right of the median line. A clinical diagnosis of gastric tumor was made and surgical intervention advised.

Report of Operation (Dr. Heyd).—The liver was enlarged and presented many stellate scars over its superior surface and particularly a marked degree of inflammatory reaction near the round ligament. Apparently there was no liver substance to the left of the round ligament. On the lesser curvature of the stomach was a neoplasm 7 cm. in length extending over the anterior and the posterior walls of the stomach, with a large crater that could be palpated through the stomach wall. It suggested a chronic infiltrative ulcer, except for the fact that there were marked reddening and stippling. Operation consisted of gastric resection and Polya anastomosis. The patient died on the third day. Permission for a necropsy was refused.

Pathologic Examination.—The specimen was apparently from the prepyloric and pyloric portions of the stomach. A considerable amount of omentum was attached on the serous surface, and was bound by bandlike adhesions to the surface. Within the fat tissue three lymph nodes were felt, each measuring 10 by 5 mm. On section the lymph glands were grayish and soft. The stomach was opened at a line between the lesser and the greater curvatures. It measured approximately 100 by 100 mm. The wall of the stomach measured 10 mm. at its widest point. The submucosa was markedly thickened, edematous and white.

The mucous membrane showed almost circular ulceration, which consisted of several fused ulcers showing serpiginous, slightly elevated, red margins. The base of the ulceration was grayish and translucent. At a short distance from the circular irregular ulceration was another ulceration, very irregular in outline, which was not as deep as the other. The mucous membrane of the stomach adjacent to the ulcer appeared thickened.

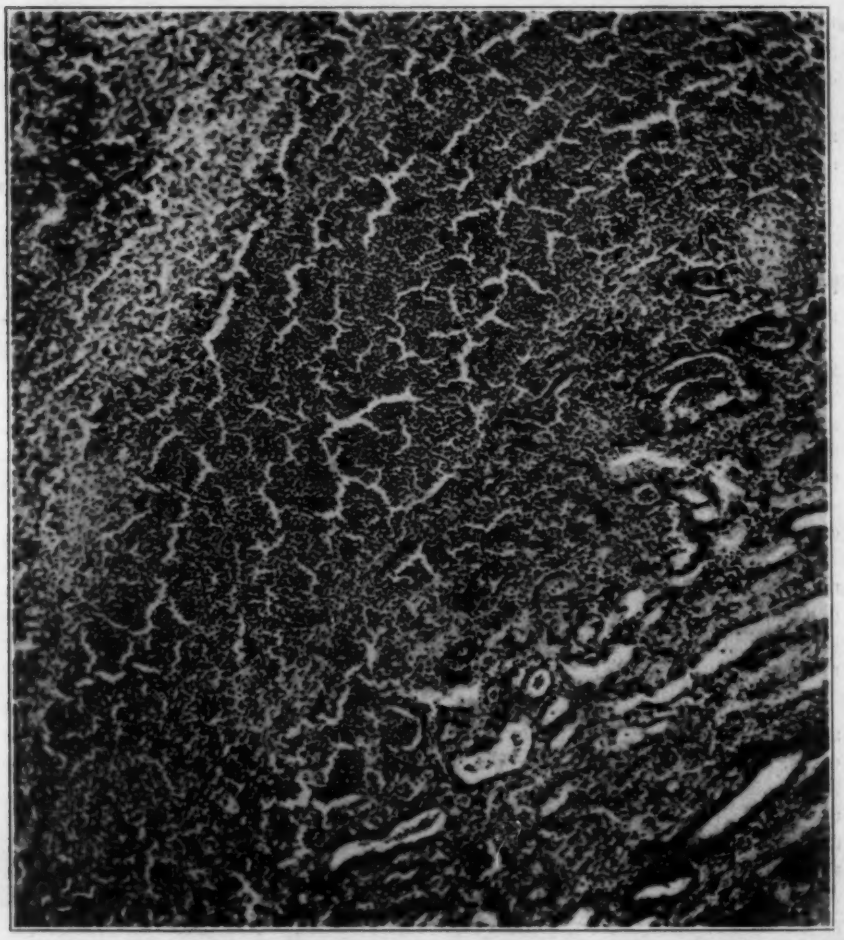

Fig. 10 (case 3).—Edge of the ulcer.

Miscroscopic Examination.—Secretions from numerous parts of the multiple ulceration revealed an almost identical picture. The mucosa and the muscularis mucosae were absent. The base of the ulcer was formed by the markedly thickened and infiltrated submucosa which was covered by necrotic tissue, invaded by numerous polymorphonuclear leukocytes and eosinophils; a fibrin network was observed in this necrotic mass. The superficial layer of the submucosa was densely infiltrated with almost exclusively polymorphonuclear leukocytes and eosinophils, with sprouting capillaries and occasional wandering

cells and proliferating fibroblasts. Sparse plasma cells were found. Several areas of necrosis were seen in this superficial part of the submucosa. The lower part of the submucosa was infiltrated by a heavy granulation tissue. However, the individual constituents were separated by edema. There were no tubercles or gumma formations. There was no perivascular arrangement of the infiltrating cells. Only occasional blood vessels in the submucosa showed some infiltration of the wall, which, however, was not marked. The lumen was only slightly narrowed and the elastic coat was without changes. The blood vessels and capillaries were crowded with numerous polymorphonuclear leukocytes.

The muscularis was thickened and edematous, invaded by numerous eosinophils and polymorphonuclear leukocytes. The blood vessels were crowded with polymorphonuclear leukocytes.

Sections from the wall of the ulcer showed similar changes. Here, however, the plasma cells became more numerous. The mucosa was intact, but in numerous places there were hemorrhages and extensive blood extravasation at the basal layer of the mucosa. The muscularis mucosae and submucosa were still thick and edematous, with abundant eosinophils and polymorphonuclear leukocytes, and some vessels showed slight perivascular infiltration of a moderate extent. This purulent granulation tissue invaded the coats of a few blood vessels, with only focal destruction of the elastic lamellae. The muscularis and subserous coat were thickened, edematous and infiltrated with various cells, with a predominance of polymorphonuclear leukocytes. The capillaries were crowded with polymorphonuclear leukocytes.

The lymph nodes showed dilated sinuses with a considerable number of proliferated endothelial cells and lymphocytes. Their architecture was normal.

Sections from the liver showed slight thickening of the Glisson's capsule, which showed infiltration with leukocytes, lymphocytes and areas of hemorrhage. The periportal fields within the liver tissue were markedly thickened and infiltrated by lymphocytes. The liver lobules were quite irregular in their appearance, were compressed and lacked a central vein. The foregoing picture led only to our diagnosis of a superficial ulcer. Because of the fact observed at operation that the left lobe of the liver was missing, a syphilitic origin of this scar was suggested but not definitely proved. Guinea-pig inoculation, culture, direct stains and stains of paraffin sections for various etiologic factors and the Levaditi method gave negative results.

The diagnosis was chronic purulent progressive ulceration, the etiology undetermined.

COMMENT

Dr. W. H. Meyer of the department of roentgenology at the New York Post-Graduate Medical School and Hospital stated that of about 18,000 gastro-intestinal roentgen-ray examinations he could be sure of the roentgenologic diagnosis of syphilis of the stomach in only five cases. In the last three years in the combined surgical services of Drs. Erdmann, Peterson and one of us (C. G. H.) of the same institution, our operatively removed material permitted us to make a diagnosis of only three undoubted cases of syphilis of the stomach. Because a patient has a positive Wassermann reaction and complains of distress in the stomach

does not justify the assumption that the condition in the stomach is necessarily due to the syphilitic infection, nor can the fact that patients with a clinical and roentgenologic diagnosis of ulcer of the stomach healed under antisyphilitic treatment be taken as establishing a syphilitic specificity to the ulcer. To us it is necessary to have unequivocal proof in one of three ways to establish the diagnosis of syphilis of the stomach. In the order of importance they may be summarized as follows: (1) roentgenologic diagnosis; (2) histopathologic diagnosis, and (3) the bacteriologic diagnosis—the finding of the spirochete in the tissues of the removed segment of stomach. It may be granted that the presence of a marked gastric deformity on roentgenologic examination and its entire disappearance under antisyphilitic treatment must be accepted *a priori* as evidence that the lesion was syphilitic. The deformity, however, must be of the nature of a distinctly deforming character, such as an hour-glass contracture, and not just the defect in luminal outline that goes with simple ulcer.

Many cases diagnosed as ulcer of the stomach occur in persons who have a positive Wassermann reaction, and simultaneously with antisyphilitic treatment the ulcer undergoes a spontaneous resolution and cure. Improvement in the general physical welfare of the patient is undoubtedly a factor in the cure of the ulcer, but the fact that an ulcer responds to treatment for ulcer in a syphilitic patient does not give us any positive data or entitle us to state that the ulceration is of syphilitic character. At the most it would be a nonsyphilitic gastric or duodenal ulcer in a man with a chronic syphilitic infection. In the histologic pictures of syphilitic lesions of the stomach we have the most intricate and complex problem. The histopathologic changes in tuberculosis and syphilis of the stomach are so close and simulate one another so frequently that it is only with the greatest nicety of judgment that some cases are placed in one or the other category. The final test, and to our minds, the crux of the situation, is the presence of the spirochete in the tissue itself. This must be an infrequent occurrence, for in McNee's case, the only one, we believe, reported up to date, after repeated sections he found only one field with spirochetes present. We feel, therefore, that a study of the cases along the three lines indicated, roentgenologically and histologically, with the attempt to detect the spirochete, is the line of study that should be assumed in all cases in which the question of diagnosis of syphilis of the stomach is concerned. It is interesting to note that in case 3 the surgeon (C. G. H), with no thought in his mind of syphilis of the stomach, noted the widespread abdominal changes and the absence of the left lobe of the liver, also the scarring and general pathologic features of the liver, criteria sufficient in his mind to suggest a chronic syphilis, but without establishing the pathologic identity of the gastric tumor. Again, in

case 1 the history of a preceding paralysis and treatment for a spinal condition is interesting as antecedent evidence in regard to the syphilitic stomach.

A consideration of the three reported cases necessitates the following questions: 1. Was the macroscopic appearance of the ulcerations sufficiently characteristic to permit an exact diagnosis? 2. What were the distinctive microscopic features on which a definite conclusion as to the nature of the lesion could be based? The points in common in all three cases are: (1) multiplicity, (2) irregularity of outline, (3) shallowness and (4) thickening of the submucosa with edema. Our three cases differed from each other in some minor features: (*a*) the nature of the margin, steep or overlapping; (*b*) the extent of the infiltration of the submucosa beyond the actual ulceration, and (*c*) the appearance of the base of the ulcer. The gross contrast to any of the common gastric lesions, such as peptic ulcer or ulcerated carcinoma, suggests primarily a diagnosis of syphilis.

The macroscopic criteria for syphilitic ulcer of the stomach, which could be compiled from a survey of the literature, are in accord with the four points mentioned in the first place. However, on microscopic examination case 3 did not exhibit anything that could let one even suspect a syphilitic lesion. Therefore, we came to the conclusion that the macroscopic picture cannot be accepted as sufficiently clear cut to permit even a preliminary diagnosis. Here we are unable to follow Sparmann,[2] who maintains that the macroscopic picture is more characteristic than the microscopic. However, the microscopic features of cases 1 and 2 were those of a syphilitic granulation tissue. The difficulty of a differential diagnosis was limited to tuberculosis or syphilis. The outstanding features in common in both cases were the diffuse and the nodular infiltration of the submucosa, with conspicuous perivascular arrangement and blood vessel changes. Aside from these points in common, however, both cases differed considerably in certain morphologic aspects. These were the origin of nodules within the lymph follicles in case 2, whereas a similar mode of development could not be demonstrated in case 1; the greater number of circumscribed nodules, Langhans' giant cells and predominance of epithelioid cells in case 2, and the presence of nodules within the regional glands and pericapsular tissue in case 2. The severity of the changes in the veins and arteries in case 1, which had produced complete obliteration of the lumen and its replacement by syphilitic granulation tissue, was not present in case 2, although periarterial and endarterial and panphlebitic changes of extensive grade were not absent. On the strength of these morphologic discrepancies, which are in accord

2. Sparmann, D.: Deutsche Ztschr. f. Chir. **164**:136 (June) 1921.

with the criteria for differential diagnosis between syphilis and tuberculosis in other organs, we strongly considered the diagnosis of syphilis in case 1 and of tuberculosis in case 2. We felt, however, that these differences cannot be accepted as full evidence on which an etiologic diagnosis can be based. In the absence of demonstrable etiologic factors (i. e., tubercle bacilli or spirochetes) we had to look for additional clinical or anatomic proofs to strengthen the morphologic evidence.

In case 1, we felt assured of our diagnosis of syphilis because of the history of chancre fifteen years previous to operation and the operative observation of a hepatic scar which impressed the unbiased surgeon as syphilitic. In case 2, however, the diagnosis of tuberculosis was sustained by the absence of any clinical evidence of syphilis, namely, the negative history and the repeated negative Wassermann reactions.

In the third case, a diagnosis of syphilis could not be made although the gross appearance was highly suspicious and the operative observation of liver changes, such as hepatic scars and the absence of the left liver lobe, were suggestive of tertiary syphilis. The lack of histologic evidence of a syphilitic granulation tissue as found in the first two cases ruled out syphilis or tuberculosis. We feel, however, that this case deserves special comment because of its peculiar macroscopic and microscopic character. The most conspicuous feature was the appearance in the gross, but microscopically also it differed from a simple peptic ulcer. The outstanding histologic features were the extensive infiltration with polymorphonuclear leukocytes; these leukocytes frequently formed small abscesses within a granulation tissue of nonsyphilitic nature. These features are those of a chronic recurrent suppurative inflammation and suggested a search for fungi as the possible etiologic agent, but this was made with negative results. We were unable to determine the nature of the unfamiliar picture since no bacteria were found and the histologic character was nonsyphilitic.

The study of these three unusual cases has led us to conclusions which primarily were applied only in our own cases. We feel, however, that these considerations might hold in other similar cases in which the gross appearance is suggestive of a syphilitic gastric lesion. It is evident that the macroscopic picture must not prejudice one in favor of a diagnosis of syphilis. In the microscopic diagnosis the greater difficulty is in the differentiation between syphilis and tuberculosis. The points of difference are not so definite that they can be accepted as absolute proof of either one or the other condition. If corroborated by clinical and anatomic evidence, however, they do permit a diagnosis one way or the other.

SUMMARY

The three cases of unusual ulceration of the stomach reported were etiologically recognized as (*a*) syphilitic, (*b*) tuberculous and (*c*) non-specific.

The conclusion was reached that, in the absence of a demonstrated etiologic factor, a correct diagnosis could be made only by the combined anatomic, histologic and clinical evidence.

DISLOCATION OF THE SEMILUNAR BONE

NEUROSPASTIC FIXATION OF THE HAND: A DEFORMITY CHARACTERISTIC OF THE INJURY *

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Dislocation of the semilunar bone, designated by some as backward displacement of the os magnum, by others as perilunar dislocation of the carpus,[1] and by still others as subtotal retrolunar subluxation,[2] is the third most frequent injury to the wrist bones. It is much more rare than Colles' fracture or fracture of the scaphoid, but its importance is second to neither if the possibility of serious permanent disability is to be considered. Grave results occur from failure to diagnose the condition and from neglect to apply proper early treatment.

At present, information in textbooks is meager and indefinite. Many contributions to the literature fail to emphasize fully the serious possibilities of permanent disability, and surgeons do not consider it as an injury of major importance.

Since the advent of workman's compensation insurance laws, and the consequent more frequent exposure of unfavorable results, this injury can no longer be considered a surgical curiosity. It is more common than is generally thought, and failure to institute early treatment can be so disastrous to the hand that every detail of its clinical characteristics should be made as common knowledge as that of Colles' fracture and other better known fractures.

It seems, therefore, that definite conclusions should be reached in regard to the management of the injury as it must be placed in the list of injuries that may produce severe loss of function.

The following eight cases selected from my own records illustrate how serious some cases may be and how widely the clinical aspects may vary. In the first case to come under my observation, case 1, the marked disability and disappointing results of treatment aroused my interest in the subject.

CASE 1.—F. N., a white laborer, aged 36, was admitted to the hospital, Jan. 4, 1921, stating that ten weeks previous to admission he had injured his wrist while cranking an automobile. Without making a roentgen-ray examination, I made a

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* Read before the Section on Orthopedic Surgery at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

1. Speed: Traumatic Injuries of the Carpus, Philadelphia, D. Appleton & Co., 1923.

2. Mouchet: Presse méd. **58**:585, 1919; Am. J. Surg. **84**: (Aug.) 1920.

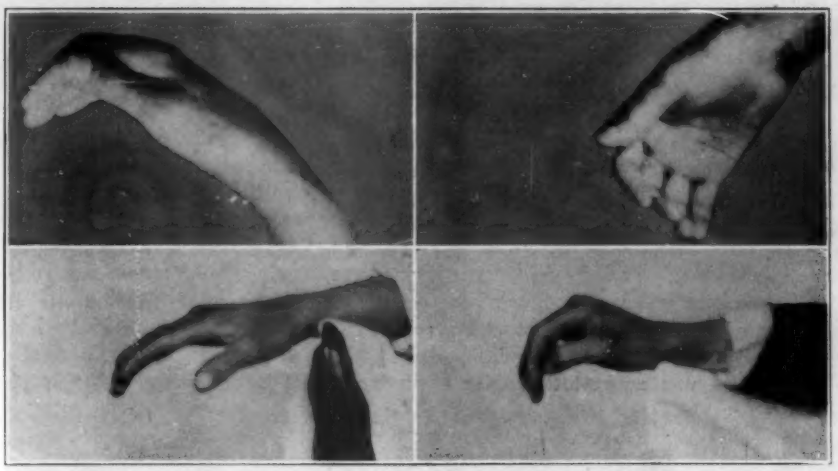

Fig. 1.—Upper part of illustration shows appearance ten weeks after injury, before operation; the thickening of the wrist and the characteristic fixed attitude of the hand should be noted. The lower part of the illustration shows the appearance two years after the removal of the semilunar bone, with limitation of motion in the fingers.

Fig. 2.—Appearance eight weeks after the injury, before operation: *A*, the semilunar bone appears thrust entirely out of its berth, lying perpendicularly in a plane anterior to the radius and the ulna. There is marked calcareous atrophy as well as rotation of the scaphoid on its transverse axis, which lifts the proximal fragment upward and backward. . *B*, conical appearance of semilunar bone in anteriorposterior view.

diagnosis of Colles' fracture. The patient said that there had been little immediate deformity, some swelling and considerable pain. An attempt was made at reduction by pulling on the hand, without giving the patient an anesthetic. A ready made anterior aluminum splint was applied, leaving free motion to the fingers, and 15 degrees of flexion to the wrist. The following day, severe pain continued, and the hypodermic injection of an analgesic was required. The roentgen-ray report was that there was no evidence of fracture. Pain of a burning, throbbing nature persisted. After three weeks, another roentgenogram was made, and the report was "forward dislocation of the semilunar bone." No attempt was made at reduction. The patient was advised that open operation would be necessary, but this was

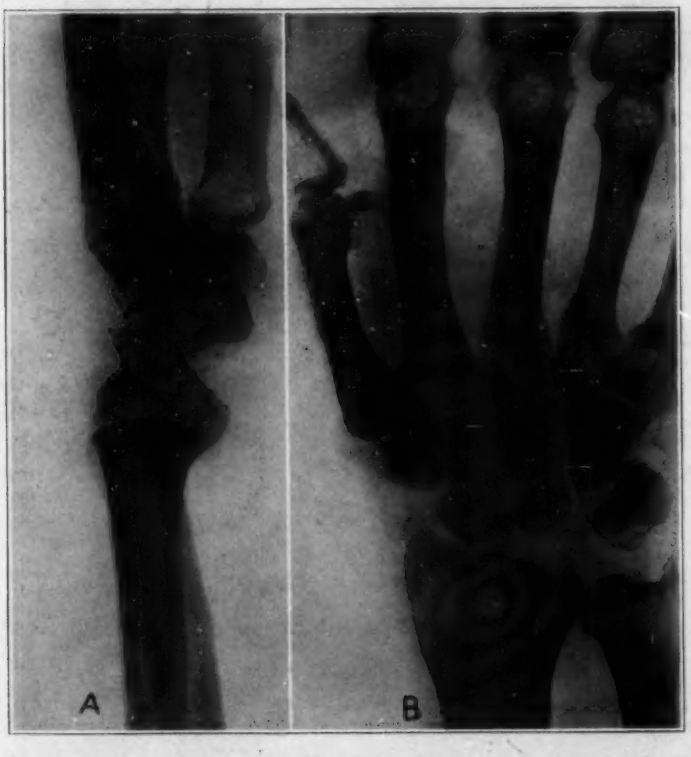

Fig. 3.—*A*, appearance after operation: The semilunar bone is missing and the head of the os magnum appears to be articulating on the radius. *B*, calcareous atrophy; the scaphoid appears short because of the rotation and the slight flexion of the hand. The space left by the semilunar bone should be noted.

delayed. On admission, ten weeks after injury, the right hand was rigidly fixed in a striking attitude (fig. 1). The wrist was flexed at 15 degrees, with no motion. There was a small amount of voluntary movement in the fingers. A slight prominence was noted at the base of the palm; the knuckles were prominent, the skin was adherent; the fingers were held slightly flexed. Roentgen-ray examination showed dislocation of the semilunar bone forward in a perpendicular position, with the concave surface facing the palmar aspect (fig. 2). There was considerable roughening of the carpal articular surfaces and evidence

of beginning rarefaction of the bone structure. The patient had had persistent pain ever since injury, for which he was given two hypodermic injections daily. There was no evidence of motor paralysis, but the patient stated that the index and middle fingers were numb. Sensation was delayed on the palmar surface of these two fingers. At operation, the semilunar bone was removed through a palmar incision made along the ulnar border of the palmaris longus tendon. Plaster was applied with the wrist in 15 degrees extension. The patient felt considerable pain for three days. On the seventh day, baking and gentle passive massage were begun, the splint being reapplied each time. Within two weeks, there was 15 degrees of wrist motion. The pain ceased rapidly after the operation, but the wrist remained sensitive to massage or activity. Improvement in the grip of the hand was slow, but the wrist joint adhesions had practically disappeared within one year. At the end of two and one-half years, the patient had full motion of wrist, but could not completely close his fist (fig. 1, lower vein). Even after this length of time, there was marked sensitiveness in the wrist, exaggerated to pain on forced movement. In spite of the effort made to prevent it, the patient became a drug addict, and died on Feb. 10, 1923.

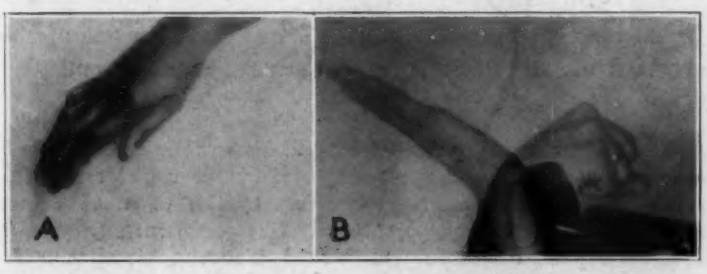

Fig. 4 (case 2).—*A*, appearance three weeks after injury, showing typical neurospastic fixation attitude; *B*, appearance showing normal function three months after open reduction.

A few months after the first patient was discharged the second patient was seen. The results in this case were surprisingly good.

CASE 2.—A white man, aged 29, a laborer in a broom factory, admitted on April 12, 1922, eleven days previously had had the full weight of a bale of broom corn rolled onto the tips of the fingers of his dorsiflexed hand, which suddenly forced the wrist into hyperextension. The patient felt considerable pain but little deformity or swelling. A diagnosis of acute sprain was made by the physician employed by the company. The pain persisted, especially on the least effort to move the wrist, and on the sixth day a roentgen-ray examination was made. Indefinite information was given to the patient and nothing was done. Persistent pain caused the patient to seek further aid.

On admission, the fingers of the right hand and wrist were held rigidly in slight flexion, the flexion at the terminal phalanges being more marked than at the knuckles. The wrist was thickened anteroposteriorly, a slight prominence being apparent at the base of the palm slightly to the ulnar side of the midline. A few degrees of voluntary motion in the fingers was possible, but a slight effort to move the wrist was painful. An attempt to straighten the fingers made the tendons in the palm prominent. The joint action was impaired, but the extent of the passive motion was little more than voluntary because of the pain and muscle

spasm. The patient complained of numbness in the fingers, especially to pin prick, and there were no motor disturbances. There was no swelling of the hand, but it seemed colder than the other and the finger tips were slightly blue. The palm was constantly moist. A deep burning sensation extended from the forearm to the fingers. There was no atrophy. Roentgen-ray examination showed forward rotation of the semilunar bone on transverse axis of about 45 degrees. No other lesion was present.

On April 12, an attempt was made at bloodless reduction and under an anesthetic but was unsuccessful.

On April 19, an open operation was performed. The semilunar displacement of the os magnum was reduced by means of a blunt probe; the semilunar bone was held in proper rotation while pressure was made on the head of the os magnum. Manipulation brought the bones into complete reduction. Splints were applied with the wrist in dorsiflexion and the fingers fully extended. The patient expressed a great sense of relief the next day. At the end of ten days, the splints were removed daily and physiotherapy treatments were given. Function returned rapidly and within two months normal motion was regained, but normal strength did not return for about four months.

A result almost as good as in case 2 was found in one of the same type in which the semilunar bone was removed on the tenth day after injury.

Case 3.—L. M., a white man, aged 28, had his wrist injured by the backfire of an engine. The bone could not be replaced by the closed method and was removed on the tenth day after the accident by Dr. C. R. G. Forrester in Chicago. I examined him two years later. His complaint was pain after he had played several sets of tennis. He said that his wrist felt as though it were sprained. The wrist and fingers appeared normal, but he had 50 per cent loss of dorsiflexion. All other motion seemed free and painless. The roentgenogram showed absence of the semilunar bone. No arthritis or disturbance of the other bones was present.

In case 4 the bone was not removed until later than in the other cases, and the patient was able to return to the occupation of a steel worker.

Case 4.—G. M., a white man, aged 21, a structural iron worker, five months previous to admission fell a distance of 20 feet, landing on his outstretched left hand. The diagnosis of a severe sprain was made and a splint was applied. The pain was severe, requiring morphine for relief. A roentgen-ray examination was not made. The splints were left on for six weeks; when they were removed, there was no motion in the fingers or wrist and the pain was still severe. In two weeks, he could move the fingers slightly. The position of the hand was: slight flexion at the wrist, the knuckles depressed and the phalangeal joints flexed more particularly at the terminal joint. All the fingers felt numb, the first and second more than the others. The hand was blue and cold. Dorsiflexion of the wrist remained painful and limited to a few degrees.

On admission Jan. 9, 1922, the wrist was thickened through the anteroposterior diameter. There was marked tenderness in the anatomic snuffbox and over the region of the semilunar bone. There was no depression on the dorsum of the wrist but fulness at the base of the palm. The finger joints were freely movable. The wrist joint had 5 degrees dorsiflexion and 10 degrees flexion. The patient could close the fist only one-third of the normal distance (fig. 9 A). The thumb

was unaffected. The hand was cold and blue and there was no tingling. Roentgen-ray examination showed the semilunar bone rotated forward on the transverse axis about 45 degrees, with the head of the os magnum in a position of subluxation back of it. There also was fracture of the posterior border of the radius, which limited the flexion and extension of the wrist. The semilunar formed an extension of the radius rather than a part of the carpal group. Reduction was impossible, because when the semilunar was freed from its adhesions it was entirely enucleated. It was also impossible to secure sufficient space for the semilunar bone between the head of os magnum and the radius. The day after the operation the patient said that it was the first time he had been free from pain since the injury. A splint was applied with the wrist in 15 degrees extension for ten days; then physiotherapy was used. Four weeks after the operation he had 10 per cent more motion in the wrist and fingers than before the operation. At the end of two months, he was able to close the fingers into the palm, and the wrist movement was about 10 degrees dorsifletion and 20 degrees palmar flexion (fig. 9 B). Nine months after operation, the patient returned to his position as a steel worker. He has full strength in his grip. There is 10 per cent loss of dorsiflexion. The other movements seem normal.

A result as good as that in case 4 was obtained in case 5, in which the bone was reduced after being neglected for three months.

CASE 5.—H. W., a man aged 21, had a heavy weight thrown on the tips of the fingers, forcing the hand backward, at the same time that the elbow was caught and held firmly. He was treated for a sprain, and splints were applied for four weeks. The pain was constant and medicine did not relieve it. On removal of the splints, the fingers could not be closed and he had little motion in the wrist joint. On admission three months after injury, his chief complaint was pain and stiffness. The wrist was thickened anteroposteriorly, and there was a prominence on the wrist at the base of the palm. There was no loss of sensation, but he stated that the middle and fourth fingers felt numb. Roentgen-ray examination showed the semilunar bone rotated forward on its axis, with the head of the os magnum back of its posterior horn. An open operation was performed with the expectation of removing the bone. A palmar incision was made; after the bone was loosened from its fibrous bed, it became possible to reduce it, and the wound was closed. Roentgen-ray examination the next day showed the bone again dislocated, in its former position. Ten days later, through a dorsal incision, the bone was again reduced; this time it was maintained by a strand of catgut hooked in a notch under the posterior horn of the semilunar bone and inserted into the dorsum of the radius. There was considerable thickening of the wrist for ten days after the operation. The stiffness decreased rapidly, but the pain, although greatly relieved at first, was persistent. The patient secured a position as a waiter three months after the operation. Six months after the operation, the contour of the hand and wrist were normal; there were 50 per cent dorsiflexion, 75 per cent palmar flexion and 75 per cent rotation. Forced dorsiflexion was painful. He had about 50 per cent of normal strength and was still improving.

Another type of case is that in which the scaphoid is fractured. The prognosis under all circumstances in such cases is poor.

CASE 6.—L. R., a white man, aged 32, a structural iron worker, was admitted on July 4, 1921, stating that six days previous to admission he had fallen 40 feet, landing on his outstretched arms and forehead. He had received a severe

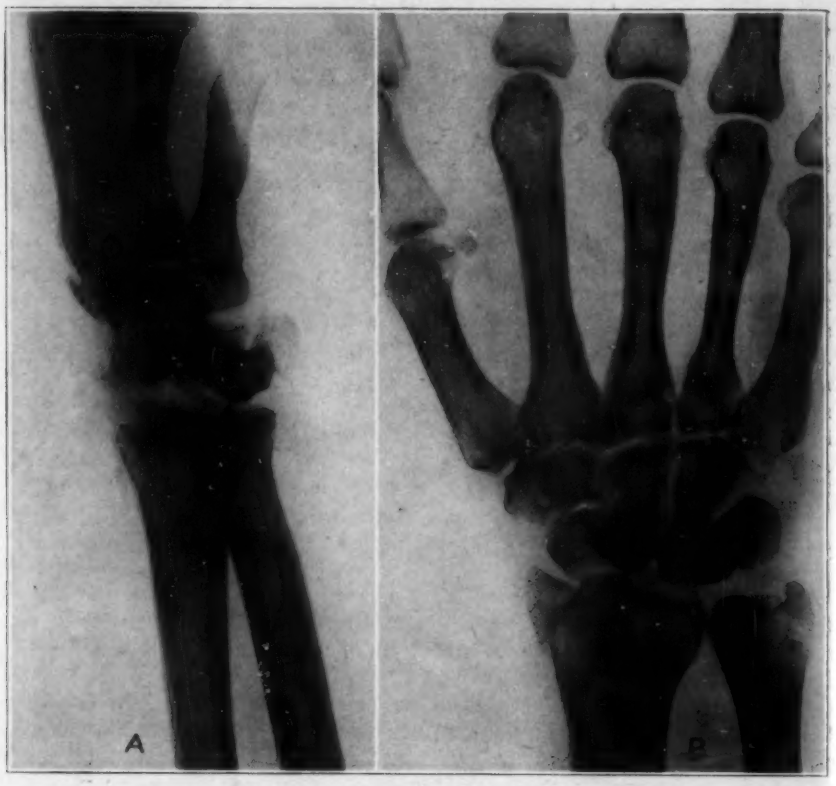

Fig. 5 (case 6).—*A*, appearance before operation, showing entire carpal section displaced backward, with semilunar and proximal fragment of scaphoid close to normal plane of radius; also numerous small fragments. *B*, fracture of scaphoid and styloid of radius; the distal fragment of the scaphoid appears as if it had forced the styloid off the radius and the os magnum is shifted outward. The conical appearance of the displaced semilunar is typical of incomplete rotation.

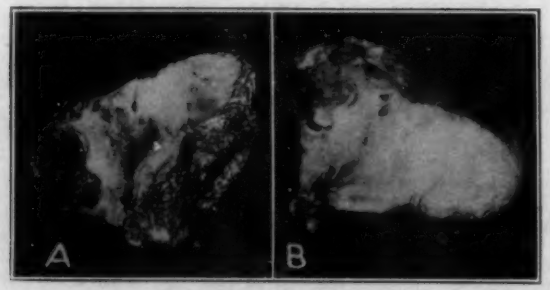

Fig. 6 (case 6).—*A*, radial articular aspects of semilunar and proximal fragment of scaphoid as they were removed with interosseous ligaments intact; *B*, carpal articular aspect of same bone.

scalp wound, dislocated and fractured the head of the radius of the left elbow, injured the left wrist and crushed the bones of the right elbow. Multiple roentgenograms were made and various injuries were cared for. A good anteroposterior view of the left wrist had been secured, but the lateral view was taken at more of an oblique angle than usual. From these views, diagnosis was made of fracture of the scaphoid and the styloid of the radius. Intense swelling of the whole arm and hand were definite early symptoms, but in spite of the many other more extensive injuries, attention frequently was called to annoying pain of an aching, burning character in the left hand. Morphine was necessary at night. Further roentgenographic examination, August 1, revealed a dislocation forward of the semilunar bone with slight displacement forward of the proximal fragment of the scapoid (fig. 5). By this time the swelling had gradually disappeared, but when the splint was removed the hand was rigidly held in a peculiar attitude as if to avoid pain. The fingers and wrist were slightly flexed, the wrist apparently was shortened and thickened; there was a slight prominence beneath the flexor tendons at the base of the palm, at which point there was marked tenderness. There was slight voluntary movement in the terminal joints of fingers, but none in the wrist. The fingers were resistent to passive motion, and an effort to force extension of the fingers or dorsiflexion of the wrist was painful. Response to a pin prick was delayed more noticeably over the tips and palmar surface of the first and second fingers, but no definite area of anesthesia or motor paralysis could be described. The palm of the hand was moist and the skin appeared glossy, but capillary circulation under the nails was good.

An operation was performed on Aug. 11, 1922. The semilunar bone could not be replaced and was removed, together with the proximal fragment of the scaphoid, through a palmar incision made along the ulnar border of the palmaris longus tendon (fig. 6 A and B). On gaining consciousness, the patient expressed satisfaction at the relief from "that annoying pain." The progress in the relief of the stiffness of the hand was slow. Immediate mobilization and physiotherapy were used. Later, traction splints were employed, and limited manipulation under ether was finally tried in order to relieve the stiffness. At the end of six months, there was considerable power between the thumb and the fingers, but it was impossible to grip an object smaller than a hen's egg. The hand appeared fixed as in fig. 1. The fingers appeared slender and the knuckles prominent. The fingers were held flexed at the terminal and middle joints, but extended at the knuckles. The skin was adherent and dry, the palm moist and the hand cold, with sensation so impaired that different coins in the pocket could not be recognized. He said that the fingers felt as though they were coated with something. In taking hold of the hand, as in shaking hands, one had the sensation of gripping an artificial hand. The corresponding elbow had practically normal movement at the end of six months, and there was no evidence that damage in the elbow might in any way be responsible for the condition in the hand.

In another case in which the scaphoid was fractured, much better function resulted.

CASE 7.—A. M., a white man, aged 35, an oil driller, on March 16, one month before admission, fell from an oil rig, landing 29 feet below on his outstretched right arm and right side. Two ribs were fractured and the right wrist was injured. The wrist was extremely painful and greatly swollen. It was treated by hot packs. On admission, April 6, the wrist was greatly swollen and the anteroposterior diameter was thickened. The fingers were slightly flexed and swollen. The knuckle joints were prominent. The wrist was slightly flexed. The

hand seemed displaced somewhat backward as a whole at the wrist joint. There was no paralysis, but he complained of numbness of the first and second fingers, and their tips burned and tingled. There was marked tenderness over the region of the wrist joint.

An operation was performed on April 8. The semilunar bone was replaced and the fracture reduced through an anterior incision. Roentgen-ray examination three days later showed recurrence of the dislocation. On April 21, semilunar and proximal fragment of the scaphoid were removed by a posterior incision. Physiotherapy was begun on May 1. Improvement was slow. The pain was greatly relieved immediately after the operation, but the stiffness was persistent.

Fig. 7 (case 8).—*A*, extent of motion after three years; *B*, prominence at base of palm; *C*, limitation of extension in wrist and fingers.

December 9, eight months after operation, he had about 50 per cent use of the hand. He could fully close the fingers but had no strength in the grip of the hand. There was about 75 per cent motion in all directions in the wrist joint, but adduction was painful. He was unable to return to his occupation.

Case 8 presents a still different picture. The injury to the carpal bones seems as severe as that in either case 6 or case 7; no operative measures were undertaken, yet the end-result was good function.

Case 8.—A white man, aged 30, a dairyman, on Aug. 17, 1919, fell 30 feet, landing on his outstretched arms. The next day he had an enormous swelling

Fig. 8 (case 8).—*A*, shortening and thickening of the wrist due to the displacement forward of the semilunar bone; rotation of the semilunar bone on its longitudinal as well as its transverse axis, obliterating the usual concave appearance; *B*, the semilunar bone, which appears trapezoid rather than conical; the proximal fragment of the scaphoid is superimposed on the head of the os magnum.

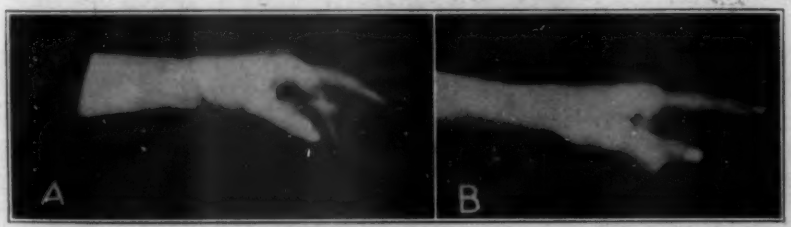

Fig. 9 (case 4).—*A*, appearance before replacement of dislocated semilunar bone, showing the extent of motion in the fingers five months after injury. The thickening of the wrist and the prominence at the base of the palm should be noted. There is full extension at the knuckles but the phalangeal joints cannot be fully extended. Flexion is practically impossible in the phalanges, but is free at the knuckles. *B*, appearance eight weeks after the removal, showing the marked improvement in motion.

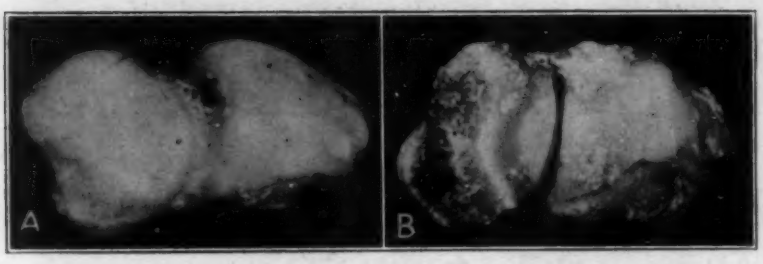

Fig. 10 (case 4).—*A*, radial articular aspect of semilunar bone, showing roughening due to firm fibrous attachment of radius; *B*, smooth carpal articular aspect.

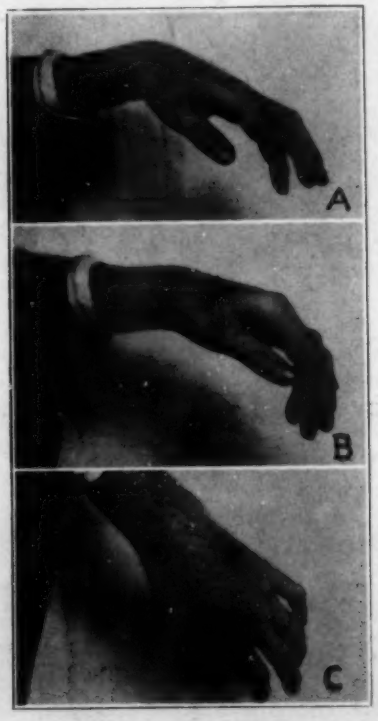

Fig. 11 (case 6).—Appearance three months after the removal of the semilunar bone and fragment of the scaphoid, showing the permanent type neurospastic fixation attitude: *A*, extent of ability to extend fingers; *B*, extent of flexion, and *C*, dorsal view, showing fork prong appearance of fingers and attitude characteristic of neurospastic fixation.

on the back of the wrist and a small symmetrical enlargement at the base of the palm. The pain was severe and morphine was required to relieve it. The tips of the fingers were "drawn" into a flexed position but the knuckle joints were not flexed. When an attempt was made to straighten the fingers, the tendons became prominent in the palm of the hand. There was slight voluntary motion in the fingers, but effort was painful. Roentgen-ray examination was made, but the diagnosis was uncertain. The consulting physicians disagreed, some stating that the wrist bones were crushed, others that there was a fracture of the scaphoid bone. The arm was placed in a splint for three weeks but the pain was continuous and the patient was given hypodermic injections daily for relief. When the splints were removed, the hand was greatly stiffened and had slight voluntary movement. The fingers were numb, especially the first and second, but there was little or no swelling. He treated and massaged his own hand, but after nine months he could not grip a lead pencil tight enough to hold it. The pain was annoying during all this time, and even at the end of two years the chief complaint was pain and weakness.

On admission, June 1, 1921, roentgen-ray examination showed dislocation of the semilunar bone and fracture of scaphoid, with a great amount of calcareous atrophy (fig. 8). An open operation and removal of the displaced parts was advised but refused. At this time, there were no adhesions in the fingers, but grip was weak and the wrist movement was limited to 50 per cent of normal. There was a large prominence at the base of the palm and a slight bony prominence on the back of the wrist in the midline (fig. 7). When pressure was made at the base of the palm, a tingling sensation was produced in the fingers and forced extension at the wrist was painful. The patient made his living dairying and was advised to use his hand for milking. On examination, March 1, 1923, practically normal function had returned, but the prominence was still present at the base of the palm and pressure at this point produced a tingling sensation. The wrist was not as strong as normal, and he sometimes dropped things easily. Roentgen-ray examination showed the bones to be in about the same condition as on June 1, 1921, except that calcareous atrophy had disappeared in all the bones except the scaphoid fragment and the semilunar bone.

NEUROSPASTIC FIXATION ATTITUDE

Each of these eight cases presents a different phase of the subject, yet there are many points common to all which may be considered characteristic of the injury.

In the first place, there is a common attitude and appearance of the hand, wrist and fingers in all cases. The illustrations accompanying the articles in the literature also show the same characteristics. Several authors speak of "a slight fork deformity" and others mention a "claw-like hand." In the semilunar cases, however, the surgeon is dealing with an attitude based on physiologic disturbances rather than on a deformity due to anatomic changes, as in the case of Colles' fracture. At first the attitude is of a spastic nature. Within one or two weeks, the attitude may become fixed, and as time goes on without treatment, this fixation may become more or less permanent stiffness with progressive degenerative changes. For convenience and as a descriptive term, therefore, I suggest the attitude be designated as that of "neurospastic fixation" of the hand.

At first the fingers are flexed slightly more at the terminal than at the middle phalangeal joints, while the knuckle joints are only a bit flexed or even fully extended or depressed; there is a fulness at the base of the palm and an increase in anterior-posterior thickness of the wrist, which may be mistaken for the deformity of Colles' fracture; examination, however, reveals that this thickness is in the middle of the wrist and not about the radius.

The fingers can be slightly flexed voluntarily, but closing of the fist is impossible. It can usually be closed by passive motion; however, during the first two weeks voluntary extension of the fingers is impossible, and an attempt to extend the fingers or to dorsiflex the wrist by passive motion is extremely painful.

In many cases the spasticity remains mild or gradually relaxes, but often within two or three weeks the pain and stiffness become extremely severe, which gives a definite appearance to the hand. Fixation of the original spastic attitude gradually takes place. The hand becomes somewhat mummy-like in appearance; there is a fixed attitude of slender pronglike fingers and a slightly flexed wrist. To grip the hand gives a cold, stiff feeling as though one had taken the hand of a cadaver. Codman and Chase, in describing one of their cases, say that "the hand looks as though it belongs to another person." The palm is moist, the knuckles are prominent, the skin is glossy and firmly adherent to the subdermal tissue. The fingers cannot be extended fully by passive force, and flexion is limited to a few degrees. Manipulation seems more like the bending of solid rubber fingers than gliding of joints.

Because of atrophy, the fingers seem slender and are separated at their bases, which gives them a fork prong appearance. There is a slight prominence at the base of the palm. The thenar eminence is somewhat atrophied, and the thumb is held close to the forefinger. Atrophy extends up the forearm, rotation being considerably limited.

The outstanding subjective symptom from the beginning is persistent pain of a burning, pressing nature, unrelieved by splints or the position of the hand. Other symptoms are general numbness of the fingers and palm, and sometimes there is a tingling sensation in the first and second fingers on movement of the wrist. The feeling in the fingers is compared by the patients to that of a hand covered with a glove.

I believe that this phenomena is a combination of nerve irritation, joint tension and bone block. As seen in case 1, it may occur in simple dislocation as well as in that complicated by fracture of the scaphoid.

PROGNOSIS AND TREATMENT

Various authors seem to differ widely in their reports. Some warn of a grave prognosis; others say nothing of serious possibilities. Some report cases in which late reduction is satisfactory and describe special

instruments for accomplishment of the operation, yet others assert that late reduction is not rational because of traumatic arthritis and absorption of bone structure, due to nutritional disturbances.

One reason for such a divergence of opinion on the part of those who have reported cases is no doubt that the clinical characteristics vary a great deal in respect to the severity of nerve and joint complications; another is that only a few cases are seen in any private practice.

As an attempt to formulate more reliable conclusions on which to base prognosis and treatment, I have made a summary of cases in the literature in which sufficient data were recorded to compare methods of treatment, results and opinions of authors.

The comparison is made from ninety-four cases recorded by twenty-nine different authors. Of this number, twenty-six were cases of closed reduction, seven of open reduction, forty-nine of early removal, five of late removal and seven cases in which the patient was untreated.

CLOSED REDUCTION, TWENTY-SIX CASES

1. *Time After Injury.*—The dislocations were reduced in nearly all cases within two or three days after injury. One was successful at four weeks. Three were complicated by a fractured scaphoid, two of which were reduced within forty-eight hours and the other at five days.

2. *Results.*—All patients report a complete return of function.

3. *Opinion of Authors.*—All agree that reduction can be accomplished best within a few hours after injury. Codman and Chase advise an attempt at a closed reduction as late as four weeks.

OPEN REDUCTION, SEVEN CASES

1. *Time After Injury.*—The shortest time after injury at which reduction was performed was six days. In the other cases, it was done from three weeks to three months later.

2. *Results.*—In three cases good function was obtained. Four patients had persistent pain and impairment of function. One of these had a fractured scaphoid; another had severe pain. The neurospastic fixation was definite, and three operations were performed.

3. *Opinions.*—Adams[3] says: "danger of traumatic arthritis, end results unsatisfactory." Codman[4] and Chase: "If closed reduction fails, try open." Knapp[5]: "Late reduction successful." Speed[1]: "Reduction after six days not advisable because of absorptive changes and traumatic arthritis." Davis[6]: "Can reduce even late cases with special skill."

EARLY REMOVAL OF BONE AND REMOVAL IN CASES IN WHICH NEUROSPASTIC FIXATION IS NOT OF A PERMANENT NATURE, FORTY-NINE CASES

Twenty of the forty-nine cases are those of Speed. The time after the injury ranged from six days to nine months. The scaphoid was fractured in several cases and dislocated in one. Removal of the whole scaphoid and the semilunar bone was mentioned in six cases.

Results.—The earlier the removal the better the results reported. No one reports perfect return of function. There were excellent results in one case in which removal was done after nine months. Several patients report satisfactory

function. When the whole of the fractured scaphoid and semilunar bone were removed, the results were reported as excellent, satisfactory, poor and improvement of the patient.

Opinions.—Adams [3] advises: "Removal where limitation of flexion and extension would interfere with work." Magnuson [7] says: "Removal gives extremely satisfactory results." Codman [4] and Chase: "If one cannot reduce, excise. Best to excise proximal half if scaphoid is fractured." Cohen [8]: "Removal gives good function." Knapp [5]: "Excision is last resort and is practically obsolete." Speed [1] advises: "Removal if unreduced at one week. If scaphoid is fractured, removal of whole bone." Stern [9]: "If one can't reduce, remove as early as possible."

LATE REMOVAL OF BONE IN CASES IN WHICH FIXATION AND DEGENERATIVE CHANGES ARE MARKED, SIX CASES

Time After Injury.—The time after injury may be from ten weeks to six years.

Result.—Elimination of pain was mentioned in two cases; otherwise, little or no improvement was noted.

Opinions.—Codman and Chase [4] say that removal is indicated for the relief of pain. This does not improve stiffness. Speed [1] says that removal eliminates pain.

CASES IN WHICH TREATMENT WAS NOT GIVEN

Results.—In every case a severe permanent loss of function is reported. One patient had pain, stiffness and numbness at six years; one could play polo after four years, but had 50 per cent limitation of wrist motion. All had pain for many months.

Opinions.—Adams [3] says: "May leave alone if good flexion or extension and if articulation with os magnum head is not displaced." Codman and Chase [4]: "Removal should have been done in each of our cases." Kleinberg [10] states: "Left alone because improvement seemed progressing satisfactorily."

SUMMARY

The experience in my own cases and in those recorded in the literature leads to the following conclusions:

A. Simple Dislocation of the Semilunar.—1. Reduction can usually be accomplished by manipulation within the first three or four days, and complete return of function can be expected.

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3. Adams, J. D.: Displacement of Semilunar Carpal Bone, J. Bone & Joint Surg. **7**:665-681 (July) 1925.

4. Codman and Chase: Ann. Surg. **51**:863 (March-June) 1905.

5. Knapp, H. B.: Dislocation of Semilunar Carpal Bone, J. A. M. A. **79**:1192 (Dec. 9) 1922.

6. Davis: Treatment of Dislocation of Semilunar Carpal Bone, Surg. Gynec. Obst. **37**:225-229 (Aug.) 1925.

7. Magnuson, P. B.: Internat. Clin., Philadelphia **3**:124-127, series 30, 1920.

8. Cohen: Ann. Surg. **73**:621 (May) 1921.

9. Stern, W. G.: Dislocations of the Carpal Semilunar Bone, J. A. M. A. **75**: 1389 (Nov. 20) 1920.

10. Kleinberg, S.: Dislocation of Carpal Scaphoid and Semilunar Bones, J. A. M. A. **74**:312 (Jan. 31) 1920.

2. In cases in which removal of bone is done within two or three weeks or in which there is mild neurospastic fixation, good function will result, but at least some permanent weakness will remain.

3. Open reduction under the same circumstances promises as good a return of function as removal. There is, danger, however, of traumatic arthritis.

4. Untreated patients have from 25 to 75 per cent permanent disability.

5. Open reduction after several weeks or in cases in which neurospastic fixation is severe should not be attempted, even though in many instances it can be accomplished. Removal is better.

6. Removal in late cases with severe fixation and degenerative changes usually will relieve the pain to a considerable degree. Improvement in function is slight.

B. Fractures of the Scaphoid.—1. Severe traumatic arthritis is so likely to occur in these cases that closed or open reduction is dangerous. If not accomplished within a few hours after injury, excision is therefore indicated.

2. Removal of the semilunar and whole scaphoid bone seems to give as good results as those secured in cases in which the proximal fragment only is removed and is the method of choice.

CORRECTION

Dr. Gabriel Tucker asks that attention be called to the following corrections in his article, "Technic of Bronchoscopic Introduction of Bismuth Subcarbonate and Iodized Oil 40 Per Cent, for Pneumonography" (Arch. Surg., January, Part II, 1927):

The reference at the end of the first paragraph, page 175, should be to figs. 5 and 6.

The reference in the fourth line from the bottom on page 181 should be to fig. 4, instead of to fig. 6.

The instrument illustrated at the top of page 182 was devised by the author, Dr. Tucker, and mention of this should be included in the legend to fig. 7.

Dr. Tucker also calls attention to the fact that the stenographic report of his discussion on the papers by Drs. Singer, Tucker, Ballon, Stewart and Archibald (Arch. Surg., January, 1927, page 216) is incorrect, in that the negative was omitted; the twelfth sentence in the paragraph should read: "In our clinics, we do not use the bronchoscope in tuberculosis unless there are special indications."

THE CAUSE OF POSTOPERATIVE RUPTURE OF ABDOMINAL INCISIONS *

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There are few things more disturbing than the postoperative rupture of an abdominal incision. This unusual disaster does not occur for a number of days, perhaps a week or more, following an operation; often when everything appears to be progressing favorably and the safety of the patient seems assured. The subject is frequently mentioned in surgical writings, but I am not aware that a satisfactory explanation has been given. The usual ways of accounting for the phenomenon are:

1. *Giving Way of the Sutures.*—This may occur by breaking, cutting through, or too early absorption. This seems likely at first thought; but when one remembers the thousands of instances in which all sorts of sutures are used in all sorts of ways without trouble, the explanation seems inadequate.

2. *Unusual Strains.*—These may be caused by vomiting, coughing or distention. Such strains, however, are always occurring, and yet rupture of an incision is rare.

3. *Infection of the Wound.*—This not only fails to explain the numerous instances of rupture without infection, but it also does not take into account the cases in which suppuration occurs, perhaps with extensive sloughing, and yet the wound remains intact, even under great strain. Furthermore, suppurating wounds not infrequently are packed with gauze, no sutures being used except in the peritoneum, without trouble resulting; and there are even those who do not use gauze at all, leaving the external wound wide open—in fact, something like this occurs when deep suppuration takes place, separating the muscles and fascia and leaving only the peritoneum below and the skin above the resulting abscess.

4. *Failure of the Healing Process (Predisposition," Age, Disease, Etc.).*—This seems to be rather begging the question, because ruptures often occur in healthy patients whose healing powers are unquestioned; and it often occurs in part of the wound while the remainder unites perfectly. And again, when a ruptured wound is reunited, healing frequently takes place without difficulty.

Recently, Weber [1] has attempted a physicochemical explanation by assuming that the underlying cause of rupture is an increase in the

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* Read in part at a meeting of the Western Surgical Association, Dec. 19, 1925.

1. Weber, M.: Zentralbl. f. chir., Jan. 3, 1926, p. 277.

acidity of the tissues, due to some form of systemic intoxication. Even if it were admitted that this might explain the ruptures that occur in connection with ileus and malignant tumors (which he emphasizes) it would afford no solution for the numerous instances in which no general intoxication exists, or in which the rupture occurs in a portion of the wound only. As he admits, this theory necessitates, in order to explain the prompt healing that occurs following secondary suture, the rather unsatisfactory assumptions that the acidity has been overcome, or that substances are generated in the blood which favor the healing process (Epstein), or that in some way the primary wound has exerted an influence on the sympathetic nervous system which favors the secondary healing (Dschanelidge).

5. *The Formation of a Hematoma.*—This might weaken a wound and increase the danger of rupture, but its expansile pressure could hardly be great enough, arising as it does from small vessels, to separate the firmly united fascia and peritoneum. In order to cause trouble, the hematoma would have to be large; but I have never seen evidences of even a small one in a ruptured wound, although a certain amount of bloody serum often is present.

While any one or several of the things mentioned may play a part in the giving way of abdominal incisions, they often do not appear to be the main factors. In many instances I believe the primary cause to be inadequate closure of the peritoneum, leaving a gap in the suture line. Through this opening the omentum forces itself, perhaps between the muscles, and even at times through the overlying fascia, which likewise may be imperfectly united. This omental protrusion not only interferes with immediate union of the wound, but as it becomes congested and swollen by strangulation it acts as a sort of expanding wedge, the effects of which are augmented by an accompanying serous exudation, the ultimate result being separation of the tissues and weakening of the incision. Any sudden strain, from coughing, sneezing or vomiting, may now precipitate a rupture, especially if infection has complicated the situation and sufficient time has elapsed to permit of partial absorption of the catgut sutures. Occasionally, as I have seen, it may be the bowel that insinuates itself between the stitches, or perhaps bowel and omentum together.

It is evident that the acceptance of this explanation would mean that the foundation for the rupture of an incision is laid immediately following an operation, and that there is a gradual increase of the trouble until disaster results, provided a number of more or less important factors are coincidentally favorable.

The foregoing hypothesis is supported by various observations:

1. Ruptures of incisions are more often seen in the central and lower part of the abdomen, where the omentum is most in evidence, rather than in the extreme upper part of the abdomen, although the strain of vomiting and distention is equally great in both situations.

2. I have always found a tongue of omentum in the ruptured wounds which I have seen, and its swollen condition and adhesions indicated that prolapse probably occurred immediately following the operation and not at the time of rupture. As other surgeons have told me of similar observations, I am convinced that it is a common experience.

3. There is often evidence that the beginning of a rupture occurs quite soon after the operation, such as tumefaction, the leakage of bloody serum between the stitches, or the presence of unusual discomfort, with nausea, vomiting, distention, and partial or complete intestinal obstruction. Such occurrences probably are produced by the incarceration of a tongue of omentum, or a portion of bowel, followed by swelling and serous exudation.

4. Every surgeon has observed how the omentum tends to protrude through a stab wound of the abdomen, and with what exasperating pertinacity it crawls between the stitches in closing a laparotomy incision, and one readily can realize how this tendency would be augmented by postoperative vomiting and distention; in fact, I have seen such a protrusion occur from vomiting before the external wound was closed.

5. The certainty with which ruptured wounds may be reunited, although the suturing may be imperfect and the wound edges apparently in poor condition for healing. This is due, I believe, to the fact that the swollen and internally adherent omentum can no longer enter the wound.

If, then, we assume that postoperative ruptures occur from within outward, and not from without inward, and that the wedging of the omentum through a gap in the peritoneal suture line is an important factor, it is evident that the danger can be lessened by greater care in closing the peritoneum. If interrupted sutures are used they should be placed very close together. If a running stitch is employed, the spacing should be short, it should be pulled tight and frequently back-stitched and locked. Ordinary over-and-over sutures are often hastily and loosely inserted, sometimes with gaps of as much as half an inch (1.2 cm.) or more. Their inadequacy may be demonstrated during an operation by passing a finger beneath the partially closed incision, especially if the suture line is relaxed by bending the patient's thorax forward—a position that frequently is assumed in bed.

Although complete ruptures of abdominal incisions are rare, subcutaneous ruptures are not uncommon. By these I mean instances in which, although the skin remains united, there is failure of union of

Fig. 1.—Abdominal incision in a dog: Separating the muscles is a partially strangulated and swollen plug of omentum, above which may be seen a cavity which was filled with serous exudate. Although the fascia had been united above the omentum, it is evident that its union has been interfered with and the strength of the wound seriously impaired. (Prof. R. G. Mills, Pathologic Laboratory, University of Colorado School of Medicine, Experiment 13.)

Fig. 2.—Plug of omentum lying in a cystlike cavity originally filled with fluid. (Prof. R. G. Mills, Pathologic Laboratory, University of Colorado School of Medicine, Experiment 14.)

either the peritoneum or of the peritoneum plus the muscles, or of both of these plus the overlying fascia. The omentum, I believe, plays just as important a part in these occurrences as it does in complete rupture, which accounts for the firm omental adhesions so frequently found in secondary operations. If the muscles, and particularly the fascia, are involved, a ventral hernia may result, in close relation to which adherent omentum usually is found, as I am sure most surgeons have observed.

This, of course, does not mean that hernias may not also be due to other causes, such as the sloughing of fascia.

Recently some experiments on dogs have been conducted for me in the department of pathology of the University of Colorado School of Medicine by J. W. Jackson and W. C. Black, under the direction of Prof. Ralph G. Mills, who will present in a separate article a more detailed account of his findings and deductions. Fifteen laparotomies were done, the incisions being closed in the usual manner, in layers, with chromic catgut throughout. In each instance a tongue of omentum was brought into the wound between the muscles, but beneath the united fascia.

In a number of dogs, especially when the amount of omentum was large and partly or completely strangulated, there was a failure of union, not only of the muscles, but also of the overlying fascia. This was due largely to the greatly swollen omentum, but also to the presence of a considerable quantity of thick lymphlike fluid, which formed a sort of cyst. Owing partly to the presence of this fluid, which I believe to be a factor, nonunion and separation of the tissues existed for some distance on either side of the omental plug (figs. 1 and 2).

It also was noticed that when strangulation of the omentum occurred, infection and suppuration were more likely to result, which increased the tendency to separation of the tissues.

Although no actual wound ruptures occurred in this series of experiments, the stage was apparently set for such an event, which might easily have taken place if the proper strain could have been produced at the proper time. It also was quite evident that the conditions often were favorable to the subsequent production of ventral hernia from subcutaneous wound rupture.

SUMMARY

As an explanation for the postoperative rupture of abdominal wounds, the hypothesis is offered that it often results from an inadequate closure of the peritoneum. Between the loose stitches the omentum (or occasionally the bowel) forces itself, and in swelling acts as an expanding wedge which forces the tissues apart and prevents proper union, the process being aided by serous exudation or, perhaps, suppuration. A rupture may then be precipitated by the strain of vomiting or distention, especially when the fascia is involved. Massive adhesions and many postoperative hernias are similarly explained by subcutaneous wound rupture, which is much more common than complete rupture.

THIRTY-FIRST REPORT OF PROGRESS IN ORTHOPEDIC SURGERY *

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(Continued from page 164)

RESEARCH

Physiology of Synovial Fluid.—Pemberton [37] and his group conclude from their experiments that diffusible substances pass easily from the blood to the synovial fluid, and that the gastro-intestinal tract must be regarded as being in surprisingly close communion with the joint fluids.

Interstitial Growth in Growing Long Bones.—Haas [38] has performed experiments in growth, using rabbits in which the measurements were controlled by being taken on roentgenograms at the time of operation and at the end of the period of observation. With a constant film target distance there is practically no chance for error, as is the case in making measurements at the time of operation. He concludes that interstitial proliferation of osseous tissue plays no part in the elongation of bone, either in the mature bone or in the young osteoid tissue bordering on the epiphyseal cartilage. Length of bone is entirely dependent on the purposeful multiplication of the cartilage cells of the epiphyseal plate.

Bone and Blood Circulation.—Mueller [39] has examined bones taken from old people with severe arteriosclerosis to determine the connection of the circulation with the nutrition of the bone tissue. Those lamellae which are near the vessels are intact, while those farther away are generally necrotic. Therefore, the lamellae in the marrow and in the cancellous bone are living, while in the compact bone only those lamellae are intact which are close to the haversian canals. In severe

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* This Report of Progress is based on a review of 230 articles selected from 595 titles dealing with orthopedic surgery appearing in medical literature between March 13 and July 24, 1926. Only the articles that seem to represent progress have been selected for note and comment.

37. Cajori, F. A.; Crouter, G. Y., and Pemberton, R.: Arch. Int. Med. **37**:92 (Jan. 15) 1926.

38. Haas, S. L.: Arch. Surg. **12**:887 (April) 1926

39. Mueller, W.: Zentralbl. f. Chir. **54**:1412 (May 29) 1926.

cases, all bone cells of the compact bone were necrotic, while the cancellous tissue still showed many living cells. He explains these observations by the thickening of the intercellular substance which, with decreasing circulation, offers an unsurmountable resistance to the circulation. These conditions are similar to those in transplanted bone and in compact bone tumors; similar changes may be seen also in certain pathologic conditions.

Avitaminosis and Healing of Fractures.—Israel [40] has made experiments in order to study the influence of scorbutus on the healing of fractures. Substituting carrots in the food of rabbits by citric acid of various concentrations, he found a reduction of the formation of callus in direct proportion to the dilution, and when citric acid was not given no callus was seen. Vitamins were withdrawn at a time when a definite amount of callus had already formed; callus continued to grow at first, but during the tenth week, spontaneous fracture took place. This in turn healed when a diet rich in vitamins was given. Similar delay in union of the fractures seems to take place in severe general infection, tuberculosis and also in pregnancy. Bier states that in cases of poor union of fractures he gives the patient half a pound of unpeeled carrots, orange juice salad and one-eighth pound of honey. He recalls a remark of Billroth, that pseudarthrosis generally heals after the patients have been removed to their homes, indicating the difference in vitamins in hospital and in home diet in Germany.

BONE, JOINT AND TENDON SURGERY

Tendon Surgery.—Harmer [41] reports a series of cases of tendon and nerve repair. The group includes 112 cases, involving chiefly the hands and wrists. The article emphasizes the principle of immediate active motion of sutured tendons without splinting. On the day of the operation, when sufficiently recovered from the anesthetic, the patient is personally instructed in exercises to move each phalanx of each finger. He is told to repeat these exercises every hour, and at some time every day he is personally supervised in these movements by the surgeon. The results as shown by photographs are excellent.

Mayer,[42] speaking of traumatic division of the extensor tendons in which primary suture of the tendons is contraindicated because of infection or extensive trauma to the adjacent tissues, states that with suitable technic tendon transplantation can be performed successfully at a later date. The tendon of the extensor communis digitorum of the index finger is the one that can be used most easily for transplantation. He lays much emphasis on the use of a special kind of tendon stitch. He

40. Israel, A.: Zentralbl. f. Chir. **53**:1414 (May 29) 1926.
41. Harmer, T. W.: Boston M. & S. J. **194**:739 (April 22) 1926.
42. Mayer, Leo: J. Bone & Joint Surg. **8**:383 (April) 1926.

splints the finger for eight days, and only at the end of this time does he begin gentle active motion. As a rule, about 75 per cent of the normal motion is recovered within four weeks after the operation.

Närvi [43] has made a study of tendon suture and tendon repair, using cats as experimental animals. His conclusions are: 1. After tendon suture the ends of the tendon are united by means of granulation tissue, which is formed by the connective tissue elements of the tendon. The ends remain passive and do not seem to form any new tissue. The connective tissue outside the tendon forms more of the granulation tissue between the ends than the peritenoneum and is therefore of more importance for the healing of the tendon wound. In the endothelium covered sheath of the tendon, the peritenoneum grows over the ends of the tendon, forming stumps that do not unite, but if sutures are passed through the cut surfaces granulation tissue is formed from the peritenoneum along and around the sutures. The author therefore recommends this type of tendon suturing. 2. The synovial fluid does not affect the regeneration of the tendon disadvantageously. The form and structure of the cicatrix of the tendon is consequently quite the same inside and outside the sheath of the tendon. 3. For restoration of function it is absolutely necessary to spare the sheath of the tendon in order to facilitate the gliding movements and to keep the tendon in the right position. 4. Only by exercises, without immobilization, is it possible to prevent adhesions of the tendon.

Arthroplasty.—In arthroplastic operations, Hass [44] recommends a different method of modeling the ends of the bones from that which is commonly used. He does not try to reconstruct the normal anatomic form, but rather to shape the bones from a functional point of view. The convex articular body—e. g., the lower end of the femur—is shaped like a wedge, while the concave body—e. g., the upper end of the tibia—is modeled into the form of a shallow cavity. The idea is by this means to minimize as much as possible broad contact of the bony surfaces and thus to prevent the formation of adhesions. He reports the results in five cases in which operations were performed by this method from one to two years previously, two in the elbow, two in the knee and one in the hip. The results in the elbows are excellent, full range of motion and function scarcely differing from normal. In the knees the range of active motion is excellent, although in one case the stability is somewhat impaired. In the hip there is active flexion of 90 degrees, abduction to 60 degrees and good extension.

[ED. NOTE.—We feel that these operations might properly be called reconstructions or resections rather than arthroplasties, as the term is commonly understood.]

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43. Närvi, E. J.: Acta Chir. Scandinav. **60**:1 (March 10) 1926.
44. Hass, J.: Zentralbl. f. Chir. **52**:270 (Nov. 28) 1925.

Osteoperiosteal Grafts for Spinal Fusion.—H. and Y. Delagenière [45] have been employing osteoperiosteal grafts in fusion operations of the spine for Pott's disease with good results. The technic is relatively simple, and this is its advantage over other methods. Massive bone transplants lose their vitality, whereas osteoperiosteal grafts survive and are therefore a better aid to fusion. The operation is performed with the patient lying on the right side. The muscles are retracted laterally and the laminae exposed and denuded of periosteum. With the chisel, long strips of periosteum lined with a thin but even layer of bony tissue are cut from the internal surface of the tibia. These are laid down in the vertebral gutter, with the bony surface against the laminae and the spinous processes. The muscles are replaced, covering the grafts, and the aponeurosis is sutured. A small drain is inserted. Following the operation the patient is kept flat in bed, but without apparatus. At the end of two months, a plaster jacket is applied and the patient is allowed out of bed. Jacket fixation is maintained for about six months. Three cases of successful operation in adults are reported.

[Ed. Note.—Some of us have been employing osteoperiosteal grafts in operations for spinal fusion for several years. The Hibbs method of fusion has been used, with the osteoperiosteal graft added for additional security. The results were impressive in one such case in which a second operation had to be performed, but not because of failure of fusion. On the side where the graft had been laid there was a large thick sheet of bone, much thicker and more solid than on the other side, where no graft was used to reinforce the fusion. If the osteoperiosteal graft alone would suffice without any other fusing procedure, it would go far toward simplifying the operation and removing its risk. We consider drainage of these wounds unnecessary.]

Correction of Deformity in Bony Ankylosis of Hip Joint.—Abbott and Jostes,[46] realizing the great risk of displacing the bony fragments in attempting to correct cases of extreme deformity of the hip joint with ankylosis by the usual method of subtrochanteric osteotomy and plaster fixation, have employed a different procedure, with entirely satisfactory results. Following the osteotomy, they put the leg up with traction in a Thomas splint, not trying to obtain correction of the deformity until the end of about four weeks. At this time, the fragments are united by firm but moldable callus. Extension is then applied to the unaffected leg in a position of wide abduction to control tilting of the pelvis. Then, gradually, the contracted hip is brought down into the desired position.

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45. Delagenière, H. and Y.: Archives franco-belges de chir. **29**:21 (Jan.) 1926.

46. Abbott, L. C., and Jostes, F. A.: Surg. Gynec. Obst. **42**:274 (Feb.) 1926.

The advantages of the method are accuracy, elimination of the risk of displacing fragments and doing away with the necessity of applying a plaster cast following the operation.

Operative Treatment of Chronic Hydrops of the Knee.—Muchin [47] has tried a new procedure for the relief of chronic hydrops of the knee, and in four cases has obtained good results. The idea is to improve the drainage and absorption from such knees by excision of a piece of the capsule. The edges of the capsular defect are sewn to the subcutaneous tissues, and into the defect a small pedunculated flap of muscle and fascia is turned. The patients on whom operations were performed have been observed for more than a year, and within this period there has been no recurrence of the swelling.

[ED. NOTE.—We judge that this procedure is based on the same idea that underlies the Kondoleon operation for elephantiasis, namely, to connect the superficial lymphatics with the part that it is desired to drain. If an essential part of the plan is the persistence of the capsular defect, then the operation would appear to be illusory, since we know that such a defect is quickly closed and the synovial lining rapidly reconstituted.]

Late Results of Resections of the Knee Performed by Ollier.—Berard and Santy [48] have investigated the late results in some of Ollier's operations of resection of the knee for tuberculosis. Twenty-five cases were selected from his records at the time of his greatest surgical activity, about thirty years ago. Of these twenty-five patients, sixteen, or 64 per cent, were found to be in good health, five did not respond, and four had died, but only one of a tuberculous disease. Of the sixteen patients that were found, eleven have been examined. All have been permanently cured of their knee joint disease. No complications have been noted. One patient, who at the time of operation had lupus of the ear and signs of a pulmonary lesion, has had pleurisy. None of the other patients ever showed any new tuberculous symptoms. In all the cases but one, ankylosis, the aim of the operation, was achieved. In the one case of failure, new tuberculous lesions were detected and a second operation was performed five years after the first, with a successful result. The knees were ankylosed with about 10 or 12 degrees of flexion. The functional results were excellent, and although the patients were mostly farmers and laborers, all were capable of the same efforts as normal people. There was no difficulty in mounting stairs, only slight awkwardness in descending. The amount of shortening varied from 4

47. Muchin, M. W.: Taschlent Turkeston, abstr. in Zentralbl. f. Chir. **53**:2037 (Aug. 7) 1926.

48. Berard, L., and Santy, P.: Arch. franco-belges de chir. **28**:963 (Nov.) 1925.

to 8 cm. Lateral deviations were not observed. Unilateral varicose veins have been a late complication, seen in almost all the cases. These seem to have a double origin, the presence of numerous scars from old draining sinuses and the absence of normal muscle contraction. On the resected side, the foot was smaller and shorter by from 15 to 20 mm. The patients have been walking with the foot in equinus, and this has led to a cavus type of foot with anterior callus formation. Although the static conditions have been changed, no painful symptoms or limitation of motion were found in the hip. In three cases, the roentgen-ray examination showed a certain degree of coxa valga, these patients being 15, 14 and 20 years of age at the time of the operation.

[ED. NOTE.—In cases of tuberculous disease no results can be considered end-results until after the lapse of a considerable number of years. The foregoing report is of interest because it represents the first attempt of which we have knowledge to obtain the entire life history of a patient following operation. The study of the functional result also is valuable. An investigation along similar lines of groups of patients treated at some of the large clinics for heliotherapy is much needed. Only by this means can we reach a conclusion as to the place of operative surgery in the treatment of joint tuberculosis.]

Surgical Treatment of Flat-Foot.—In cases of severe contracted flat-foot, Meyer [49] has obtained good results by freezing the peroneal nerve for two or three minutes with ethyl chloride. The nerve is exposed above the head of the fibula. By this means the peroneal muscle group is paralyzed for a period of about six months, and during this time the feet are treated by massage, exercise and foot plates. The method has proved satisfactory in forty-nine cases, and in all the function of the nerve has been recovered in from five to seven months. Even in bilateral cases the function of the feet was so little embarrassed that the patients were able to resume their work almost immediately following the operation.

Schulze-Gocht [50] does not agree with the view that the action of the peroneus brevis is similar to that of the peroneus longus, and that both are bad mechanical factors in flat-foot. He points out that the latter muscle draws the midportion of the foot outward, but the forefoot inward, while the peroneus brevis raises the outer border of both tarsus and metatarsus. In the surgical treatment of flat-foot, the latter muscle should be eliminated while the former should be reinforced. He therefore exposes the peroneal tendons above the external malleolus, sutures the short peroneal to the long peroneal tendon and cuts the tendon of

49. Meyer, Hermann: Beitr. z. klin. Chir. **135**:150, 1925.
50. Schulze-Gocht: Arch. f. Orthop. d. Unfallchir. **24**:32, 1926.

the former distal to the point of suture. The primary results have been encouraging, although as yet sufficient time has not elapsed to know the final outcome.

In another article, the same author [51] deals with the treatment of pes cavus along similar lines. He points out that in cases of isolated paralysis of the peroneus longus muscle there results a flat, pronated foot. Consequently, in cases of hollow foot in young persons he cuts the long peroneal tendon and sutures this muscle to the peroneus brevis. In all cases in which this procedure has been employed there has been noted a progressive correction of the deformity.

FRACTURES

Muscle and Nerve Injuries Associated with Fractures.—Lewis,[52] speaking of nerve and muscle injuries associated with fractures, states that these lesions are relatively rare, but that when present they so prolong the disability that early recognition and intelligent therapy are demanded. In 80 per cent of the nerve injuries, the humerus is the bone that is fractured, the common site of the fracture being in the lower third of the bone (60 per cent). Of the peripheral nerves involved, the musculospiral accounts for 60 per cent, and the ulnar nerve for about 25 per cent. Many of the latter, however, are late paralyses. Ischemic myositis (Volkmann's contracture) is one of the most disabling lesions associated with fractures. It is observed most frequently after supracondylar fractures and fractures of the lower end of the humerus. It is caused by venous congestion. About 8 per cent of the cases of ischemic palsy occur without a cast or constricting dressing having been applied. The importance of the subfascial hematoma in the production of ischemic palsy has not been fully appreciated. A fracture should not be considered merely as a skeletal injury, but a careful examination should also be made for associated injuries of nerves, muscles and blood vessels.

[ED. NOTE.—Some of us have encountered cases in which ischemic myositis was present when the patient was first seen and before any dressing had been applied for the fracture. Contrary to the experimental evidence which is in favor of venous congestion as the causative factor, the finding in these cases was absent radial pulsation, pallor and coldness of the hand, together with inability to move the fingers. The first two developed typical ischemic contractures. In the third case, multiple incisions were made at the elbow, and part of the subfascial hematoma evacuated, with the result that the ischemia disappeared and the hand remained normal. This procedure would seem worthy of trial in any similar case.]

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51. Schulze-Gocht: Arch. f. Orthop. d. Unfallchir. **24**:19, 1926.

52. Lewis, Dean: Proc. M. Soc. State of N. Y., March 30, 1926, reported in J. A. M. A. **86**:1380 (May 1) 1926.

Age of Fractures.—In an effort to decide whether it is possible to determine the age of a fracture by its roentgenologic appearance, Andrei [53] has made a study of 269 cases of fracture of the shafts of the long bones and of the phalanges. His conclusions are as follows. 1. In simple diaphyseal fractures without displacement, the first modification of the appearance is a blunting of the margins and of the bony spicules, which becomes evident at the end of the second week. The callus begins to appear between the sixteenth and the twentieth days after the injury. It becomes clearly delineated at the third or fourth month, and acquires the opacity of a normal bone between the eighth and tenth month. During the first year, the callus remains free from lamellar structure. The reconstruction of the lamellar system begins at the thirteenth or fourteenth month, and is well advanced at the end of the second year. The line of fracture disappeared from the sixth to the eighth month after injury, and reappears later as a line of increased opacity. At the end of the second year, although there has been a conspicuous reduction in the size of the callus, there always remains a trace of it in the form of a thin, opaque layer that thickens the bone. 2. In diaphyseal fractures with displacement of the fragments the blunting of the margins and spicules of the bones appears at the end of the second week. The callus begins to be evident in the third or fourth week. It becomes delimited from the contiguous tissues later than in other types of fracture—that is, toward the sixth or seventh month—and contemporaneously regains the opacity of normal bone. It undergoes a process of reduction less than in fractures well reduced, and the reconstruction of the bony structure is not made at any fixed period. 3. In the fractures of the phalanges, of the metacarpals and of the metatarsals, a blunting of the margins and of the spicules is seen at the end of the first week. A few days later, and in general before the end of the third week (sometimes even at the beginning of the second week), callus appears in the form of a delicate white cloud. At the end of the fourth month it is clearly delimited, and toward the sixth or seventh it regains the opacity of normal bone. Even in these fractures, the callus remains free from lamellar structure during the first year after injury. This structure appears at the thirteenth or fourteenth month and is more or less advanced at the end of the second year. This line of fracture disappears between the sixth and eighth months, to become evident later in the form of a bone cicatrix.

Local Anesthesia in the Reduction of Fractures.—Cohen [54] advises the more general use of local anesthesia for the reduction of fractures. The method employed has been one of local infiltration at the site of the break, and the experience covers a period of two years, during which

53. Andrei, O.: Chir. d org. di movimento **10**:255 (Feb.) 1926.
54. Cohen, Ira: J. A. M. A. **86**:1896 (June 19) 1926.

time it has been used in all types of cases. The nerve block method, either of the brachial plexus for the upper extremity or by spinal injection for the lower extremity, has also been used in exceptional cases. The local infiltration method is easier and has proved a practical procedure. It has been used satisfactorily in fractures of the shaft of the femur, humerus and tibia. The one contraindication is a compound fracture. The muscular relaxation which is obtained is not as great as with general anesthesia.

[ED. NOTE.—Some of us have employed the nerve block method of local anesthesia, although we have had no experience with the local infiltration method. For the reduction of Colles' fracture and other types of forearm and elbow fracture in persons who are poor anesthetic risks, it is the method of choice.]

Traction on Sternum in Treatment of Multiple Fractures of the Ribs.—Jones and Richardson [55] report the case of a man with multiple fractures of the ribs, from the second to the eighth on one side, with great shock and marked difficulty in breathing. They made an incision on either side of the sternum, through which the jaws of a bullet forceps were introduced to grasp the sternum. They were locked in this position and by means of a cord fastened to the handles, leading to an overhead pulley and counterpoise weight, continuous traction was exerted on the sternum and depressed portion of the chest. The dyspnea was greatly relieved. The danger in the procedure lies in the proximity of the pleura.

Fracture of Semilunar Bone from Indirect Violence.—Palmer [56] reports an unusual case of fracture of the semilunar bone from indirect violence. The fracture was visible only in roentgenograms taken with the wrist in the position of extreme flexion. These showed the anterior horn separated from the body of the bone. The injury was produced by lifting a 100 pound bag of cement. The symptoms were pain and weakness.

Ununited Fracture of the Hip.—Henderson,[57] writing on ununited fracture of the hip, asserts that the autogenous bone graft, the aim of which is to restore as nearly as possible the normal anatomy, is the operation of choice. He asserts that the fact that success was attained in 76 per cent of the twenty-one cases in which he has performed this operation indicated that this procedure compares favorably with bone grafting operations for nonunion in other bones. In three cases the same results were achieved by using the beef bone screws, but these

55. Jones, T. B., and Richardson, E. P.: Surg. Gynec. Obst. **42**:283 (Feb.) 1926.

56. Palmer, J. H.: Canad. M. A. J. **16**:170 (Feb.) 1926.

57. Henderson, M. S.: Ann. Surg. **83**:696 (May) 1926.

cases were more favorable in every way. The reconstruction operations of Brackett and Whitman were carried out in six other cases, with good results in all. In the latter group, however, there was more residual stiffness than in the former group, and the function, although satisfactory, was by no means as good. The duration of the nonunion is no criterion in selecting the type of operation. Some of the best results from the bone grafting operation were obtained in cases in which the nonunion had existed for from two and one-half to three years. The indications for the operation are considered to be a good state of health, considerable disability and the persistence of a sufficient amount of the neck of the femur.

Fractures of the Os Calcis.—Conn [58] believes that fractures of the os calcis can be classified into five groups according to the character of the predominating deformity: (1) eversion of the calcaneum, resulting in a more mesial deflection of the weight-bearing line; (2) upward displacement of that portion of the posterior tuberosity serving as the attachment of the tendon of Achilles; (3) impactions resulting in shortening and upward displacement of the posterior tuberosity without separation of the tendon of Achilles; (4) serious distortion of the articular surfaces; (5) those cases in which by reason of the location or because of the destructive comminution, nonunion is impending or present. Conn states that fractures of the os calcis are serious and disabling injuries, in which the results continue to be "incredibly bad." Roentgenograms should be made in a manner well calculated to reveal both the perpendicular and lateral deflections. In fresh fractures, procrastination, attempts at manual deduction, and ineffectual immobilization should be abandoned in favor of the more radical and efficient skeletal traction procedures, tenotomy of the heel cord and, when indicated, subastragalar arthrodesis.

Subastragalar Arthrodesis in Treatment of Old Fractures of the Calcaneus.—Reich [59] reports four cases of old fractures of the os calcis which he treated by performing the operation of subastragalar arthrodesis. The results were satisfactory. He asserts that the pain in the old fractures is almost invariably due to a traumatic osteo-arthritis resulting from extension of the fracture into the subastragalar joint.

[ED. NOTE.—We agree that the only way by which the present poor results from fracture of the os calcis can be improved is by the more general adoption of radical procedures such as advised by Conn and Reich.]

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58. Conn, H. R.: Radiology **6**:228 (March) 1926.
59. Reich, R. S.: Surg. Gynec. Obst. **42**:420 (March) 1926.

AMPUTATIONS

Artificial Limbs: Their Relation to the Different Types of Amputation Stumps.—LeMesurier,[60] writing on amputation stumps viewed from the standpoint of prosthesis, points out that in spite of all that has been published on this subject since the war, few surgeons appear to have enough knowledge of artificial limbs to do the best thing for their patients. In civil practice, one still sees stumps that have been amputated at unsuitable levels, improperly fashioned flaps and joint contractures. There has been no general adoption of the temporary shrinkage appliances which result in such great saving of time and money and there appears to be a surprising lack of knowledge of even the fundamental principles of prosthesis. He is strongly of the opinion that the end weight bearing stumps, such as the Syme and the Stokes-Gritti, give the best results, and raises the question whether the results obtained with the latter are not even better than those with amputation of the leg below the knee. His description of the standard types of appliance to be used with the various kinds of amputation is clear, as also the exposition of the principles of their use.

Kineplastic Amputation: Arm-Bimotor and Prosthesis.—Bosch Arana [61] reports the results of his experience with the kineplastic amputation in the upper extremity, employing a bimotor made from the biceps and triceps muscles. Three factors enter into the success of the procedure: the surgical factor, the orthopedic factor and the human factor of the individual patient. The latter is the key to complete success. Discussing results, the author states that all of his patients can grasp any object of average weight, can lift it to the mouth or either side of the head and can bend the arm or extend it. They can perform all the motions of pronation and supination necessary to hold objects or to take articles and carry them to the mouth and raise the hand in complete abduction so as to form a right angle with the body.

MISCELLANEOUS

Cysts of Semilunar Cartilages.—From a study of three cases of cysts of the semilunar cartilages and from a review of cases previously reported in the literature, Allison and O'Connor [62] conclude that these cysts represent the end-result of a degenerative process caused by an interference with the blood supply to a portion of the cartilage, the exciting cause being a nonlacerating injury. The salient features common to all the cases are: 1. The cysts are multilocular. 2. They have

60. LeMesurier, A. B.: J. Bone & Joint Surg. **8**:292 (April) 1926.

61. Bosch Arana, G.: Surg. Gynec. Obst. **42**:416 (March) 1926.

62. Allison, N., and O'Connor, D. S.: Surg. Gynec. Obst. **42**:259 (Feb.) 1926.

no endothelial lining (exception, Ollerenshaw's case). 3. They are filled with a mucoid substance. 4. There is no evidence of inflammatory reaction about them. 5. The cysts have in all cases been located in the midportion of the semilunar cartilage on the external border. 6. A definite history of injury was present in almost half of the cases. 7. The cysts reach their maximum size quickly and then remain stationary. 8. Most of the patients were in the second decade of life. 9. Spontaneous recovery is unknown and recurrences have taken place in which the entire cartilage was not removed. 10. Pain is present on complete extension and on acute flexion of the knee.

[ED. NOTE.—We have been impressed with the fact that cysts of the semilunar cartilage are not uncommon and that in the majority of the cases the diagnosis can be made readily from the clinical evidence.]

Posterior Contusion of the Patella.—Haglund [63] calls attention to a lesion that has been encountered frequently in his clinic, but which apparently is not generally recognized. He terms the condition posterior contusion of the patella and ascribes it to a severe trauma to the patella when the knee is flexed. The roentgenographic appearance is said to be characteristic. In the lateral view, there is a distinct cavity in the posterior surface of the patella, while the surrounding bone shows increased density similar to that of cortical bone. In early cases, atrophy and necrosis can be made out, the picture at times somewhat resembling tuberculosis. Haglund believes that the condition recently described by Freund under the name "chondropathia of the patella" is the same lesion. Contrary to Freund, Haglund does not consider operation necessary for cure in all cases, and says that many of them heal without any special treatment.

Lesions of the Sacro-Iliac Joints.—Painter [64] has made an exhaustive survey of all the evidence that has accumulated in respect to the anatomy, physiology and pathology of the sacro-iliac joints. From this study he feels that the conclusion is warranted that the sacro-iliac articulations are subject to the same lesions as all other joints and that the presence of a pathologic condition in the joint is manifested by the same signs and symptoms that are encountered elsewhere. He feels that the evidence is overwhelming that sciatica is a cardinal symptom of sacro-iliac lesions, and that it is due to a perineuritis brought about by the proximity of the radicals making up the sciatic nerve to whatever lesion is affecting the joint. He states that the accumulated experience shows that in the traumatic lesions of the sacro-iliac joints good results may ordinarily be obtained from mechanical and physiotherapeutic methods of treatment, and that this should cause those advocating operative measures

63. Haglund, P.: Zentralbl. f. Chir. **53**:1757 (July 10) 1926.
64. Painter, C. F.: Boston M. & S. J. **194**:613 (April 8) 1926.

other than manipulative to hesitate. It is his belief that open operative interference in this group of cases is practically never indicated, and should never be undertaken until after prolonged spica treatment has been tried.

[ED. NOTE.—Painter's study and critical analysis of the evidence in favor of sacro-iliac lesions as a cause of backache is a valuable contribution and deserves to be read. It is fair to say that we are not in agreement concerning the relationship of sciatic pain to sacro-iliac lesions. While it is the part of wisdom to warn against too hasty operative intervention now that the technic of arthrodising operations has been perfected, we recognize that this procedure has a definite place in the treatment of certain of these cases.]

Periarthritis of the Shoulders.—Schulkof [65] has made a study of the periarticular spaces of the shoulder by means of the injection of iodized oil, 40 per cent, and various dyes. He finds in practically all cases a definite connection between the two large bursae, the subdeltoid and the subaxillary. In cases of periarthritis of the shoulder, he has injected from 30 to 50 cc. of 0.125 per cent solution of procaine into the subdeltoid bursa and found that by this means he was able to relieve the pain and to mobilize the shoulder.

Scapular Myalgia.—Fuerstenberg [66] believes that scapular myalgia constitutes a distinct entity. The condition begins rather suddenly with pain in the scapular region, which rapidly increases in severity. It is made worse by movement and relieved by rest. The arm seems powerless and is held rigidly to the side. The muscles of the scapula are tense and hard, showing distinct hypertonicity. There is marked tenderness on pressure and the skin is hyperesthetic. If the condition lasts, the muscles atrophy. The shoulder joint itself shows no limitation of motion. The roentgenograms are negative. He believes the condition may originate in infection from remote foci, from exposure to cold, from mechanical strain, but especially from metabolic disturbances.

[ED. NOTE.—Before making a diagnosis of scapular myalgia one must be careful among other possibilities to eliminate lesions of the spine with referred pain to the scapular region. Careful methods of examination have reduced the number of cases of myalgia to the point that this condition is now rarely diagnosed.]

Osteochondritis Dissecans.—Koenig,[67] in a discussion before a German surgical congress on osteochondritis dissecans, stated that the name osteochondritis dissecans is not sufficiently descriptive of the process, as

65. Schulkof: Zentralbl. f. Chir. **53**:1364 (May 29) 1926.
66. Fuerstenberg: München. med. Wchnschr., **73**:734 (April 30) 1926.
67. Koenig: Zentralbl. f. Chir. **53**:1411 (May 29) 1926.

the main factor is a necrosis, whereas the inflammation and dissection are secondary characteristics. It has often been questioned whether the process is of traumatic or nontraumatic origin. Koenig agrees with his father that both causes may be found. He had made a careful study of twenty-seven cases at different stages. The histologic examination showed necrosis of the bone and of the fibrous tissue within the marrow. He supports the views of Axhausen in respect to embolism as an etiologic factor in certain cases, and states that the production of osteochondritis by this means has been clearly demonstrated by experiments.

Kienbock's Disease.—Fontaine [68] regards Kienbock's disease of the carpal semilunar bone as primarily a process of atrophy following trauma. He reports the case of a young man who injured the semilunar bone by catching a brick which had been thrown to him. Atrophy and softening of the bone followed, but all disturbances ceased after the removal of the semilunar bone. He calls attention to the fact that Müller obtained a good result in a similar case by curetting the interior of the bone, preserving the outer cortical shell.

Osteochondritis Metatarsophalangeal, Infraction of the Second Metatarsal Bone, March Foot.—The lack of knowledge concerning a condition of the head of the metatarsal commonly designated by the name of Koehler is well shown by the presence of three articles in the current literature, each describing a more or less similar condition under a different name. Under the title "Osteochondritis Deformans Juvenilis, Metatarsophalangeal," Schaller and Nadaud [69] describe the roentgenologic features in a case that they have followed. There is a shortening of the head of the bone, with flattening and broadening, condensation of the spongy bone, widening of the intra-articular space and preservation of the normal appearance of the opposing articular surface. A plaster splint was applied in their case, but notwithstanding this, the lesion progressed markedly during the ensuing five months.

Freiberg [70] employs the term metatarsal infraction, and reports a case in which the first roentgenogram did not show any bony abnormality, but a second roentgenogram taken eight months later showed the typical changes mentioned by Koehler. He acknowledges that simple trauma is not a satisfactory explanation of the clinical and roentgenologic phenomena that have been described in this condition.

March foot is the term Jansen [71] applies to the well-known edematous swelling of the metatarsal region of the foot which is seen in persons who are made to walk beyond their strength. Jansen considers this the

68. Fontaine, R.: Rev. de Chir. **63**:769, 1926.
69. Schaller and Nadaud: J. de radiol. et d'électrol. **10**:123 (March) 1926.
70. Freiberg, Albert: J. Bone & Joint Surg. **8**:257 (April) 1926.
71. Jansen, Murk: J. Bone & Joint Surg. **8**:262 (April) 1926.

result of overfatigue in a weak foot, with resulting spasm of the interossei muscles, circulatory stasis, thickening of the periosteum and brittleness of the bony tissue, eventually leading to fracture.

Roentgenologic Observations in Hemophiliac Joints.—Doub and Davidson [72] classify the roentgenologic observations in hemophiliac joints in two groups: the early cases without destructive changes and the advanced cases with destructive changes. In the first group, there is simple effusion of blood, which may or may not show calcification. There is thickening of the joint capsule and lipping similar to that of hypertrophic arthritis. In the second group, there is organized blood clot in the synovial cavity, punched-out areas of bone destruction in the epiphyses and points of cartilaginous destruction in the articular surfaces.

Lateral Roentgenography of the Hip Joint.—Béclère and Porcher [73] have made a study of various possible methods of obtaining lateral roentgenograms of the hip joints. The positions used were as follows: 1. The patient lies supine, the unaffected hip being flexed 90 degrees. The rays are directed upward from below the ischial tuberosity on the opposite side, passing through the adductor muscles of the affected hip. The plate is placed in close apposition to the trochanter, and the leg is in complete extension, the patella facing upward. 2. The patient lies supine with the legs fully extended. The rays enter on the side of the hip to be examined above the trochanter, and the plate is placed against the adductor muscles, perpendicular to the table. 3. The patient lies midway between the lateral and the prone positions. The plate is placed anterior to the trochanter, and the rays are directed laterally and anteriorly through the buttock. 4. The patient lies on the side, but rolled a little backward so that the rays may enter just above the pubes, the plate being placed beneath the trochanter. 5. The patient lies supine. The rays enter above the ilium of the opposite side and pass through the body to reach the hip, the plate being placed against the trochanter and perpendicular to the table. 6. The patient lies prone at the edge of the table, the affected hip being slightly flexed with the leg extending down to the floor. The rays enter posteriorly and above the trochanter, the plate being placed anterior to the hip. 7. The patient lies supine, with the hip flexed to 90 degrees, and in a position of forced abduction. The rays are directed toward the hip from in front near the median line, the plate being placed behind. The authors state that the clearest outline of the acetabulum is obtained in the first, third, fifth and seventh positions.

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72. Doub, H. P., and Davidson, E. C.: Radiology **6**:217 (March) 1926.

73. Béclère, H., and Porcher, P.: J. de radiol. et d'électrol. **10**:97 (March) 1926.

BOOK REVIEWS

ORTHOPEDIC SURGERY. By W. B. COCHRANE. Edinburgh: E. & S. Livingstone, 1926.

Much is heard of preventive medicine, but little of preventive surgery, and yet in orthopedic surgery the ounce of prevention is often worth the ton of cure. Cochrane founds his work on orthopedic surgery on preventive measures of physical education, acknowledging his debt to his American teacher, Goldthwait. He recalls the work of Nicholas Andry, who defended his thesis "Orthopaedia" before the Faculty of Medicine in Paris, of which he was the dean. Having laid his foundations of body mechanics, Cochrane proceeds to outline under anatomic groupings the various diseases and lesions of the locomotary apparatus which come within the field of orthopedic surgery. The book is designed to give medical students an idea of the scope of this branch of surgery, and to suggest to them the principles of diagnosis and treatment. It fulfils its purpose admirably, and will be found even more valuable for general practitioners than for undergraduates. It is an original and sound presentation which should be widely read.

THE INFLAMMATION AND TOXIC DISEASES OF BONE. A TEXTBOOK FOR SENIOR STUDENTS. By R. LAWFORD KNAGGS, M.C., F.R.C.S., Consulting Surgeon to the Leeds General Infirmary; formerly Professor of Surgery in the University of Leeds. New York: William Wood & Co., 1926.

This interesting book, written by a surgeon of wide experience, who has devoted the years since his retirement from active surgical duties to research, is a valuable contribution to the subject of inflammatory and toxic diseases of the bone. As he says, it is a textbook for senior students, and the subjects that receive special attention are osteomyelitis, tuberculosis, syphilis, the arthropathies, yaws, arthritis deformans, rickets, scurvy, osteomalacia, Pott's disease, leontiasis ossea and osteogenesis imperfecta. There is a wealth of illustration of both the macroscopic and the microscopic appearance of these diseases, and the etiology, pathogenesis and morphology are extremely well covered, while only the general principles of treatment are outlined. He believes that most of the conditions that he describes have a background of infection, and that their lesions are probably caused by toxins elaborated in the body from such foci. The book, which consists of 400 pages, is a valuable contribution to the subject of inflammatory and toxic diseases of the bone.

BONE SARCOMA. By E. A. CODMAN. New York: Paul B. Hoeber, 1926.

This book describes in concise terms the uniform classification of tumors of the bone recognized by the American College of Surgeons. It will lead to a much clearer conception of tumors of the bone and will enable surgeons everywhere to compare results and observations in a common language. Fortunately, it is to be widely distributed by the American College of Surgeons, and will undoubtedly be closely followed.